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**A STUDY ON**

**KALANCHAGA PADAI**

**(DISSERTATION SUBJECT)**



*For the partial fulfillment of the  
requirements to the Degree of*

**DOCTOR OF MEDICINE (SIDDHA)**

**BRANCH III – SIRAPPU MARUTHUVAM**

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# INTRODUCTION

Man has eternally endeavoured to keep himself free from three types of therapeutic namely, physical, mental and spiritual. Therefore the history of medicine is as old as the history of mankind. According to Indian tradition, the primary objectives of human life are to perform religious rites of acquire wealth to satisfy the worldly desires and to attain salvation. A popular saying that “A sound mind in a sound body”

In addition system of medicine developed in various parts of the world during different ages.

The word siddha was derived from the word Siddhi (trans consciousness). Siddhi is interpreted in two ways. “Attaining perfection in Life” and “Heavenly bliss”. The siddha system viewed in terms of the etymological meaning can be defined as a system which can lead one to attain perfection in life and heavenly bliss.

The Siddha system, a branch of Indian systems of medicine, is holistic in its approach. The siddha medicine has its base on the principles of nature and its elements.

The siddha system is based on 96 thathuvams (philosophies). These thathuvams include five fundamentally as well as basically known as,

1. Five basic elements (Pancha boothams) namely Vin, Kaal, Anal, Punal, Mann,
2. Five senses (Pulungal) namely Saptha, Sparisa, Ruba, Rasa, Kantha.
3. Five sensory organs (Porigal) namely Mei, Vaai, Kan, Mookku, Sevi.
4. Three humours – Vaadham, Piththam, Kabham

The entire universe is also made up of Pancha boothams. So any changes in the cosmos will reflect in the human body. This was quoted in Sattamuni Gnanam by Sattamuni as follows.

”அண்டத்தில் உள்ளதே பிண்டம்  
பிண்டத்தில் உள்ளதே அண்டம்  
அண்டமும் பிண்டமும் ஒன்றே  
அறிந்து தான் பார்க்கும் போதே”

- சட்டமுனி ஞானம்

First of all Siddhars employed the practice of “Aasanas’ and ‘Praanayaamam’ for all the human beings to lead to longlife as ‘karpa’ medicinal therapy.

Siddhars classified in primarily the diseases based on signs & Symptoms.

Their centuries old researchful and enlightenment of Siddha initially deals with etiology, pathogeneis, imbalance of humor, diagnosis and investigations (Envagai Thervugal). Secondly it is not a new one to Siddha everybody easily to get knowledge of this points of view as per the siddha text book refferences. Siddhars had a thorough knowledge of therapeutic values of herbs through their intuitions. They had tried and became very well – versed in preparations by using their scientific knowledge of a alchemy.

The whole body is governed by their physilogical factors called ‘three thathoos’ namely Vaadham, Piththam and Kabham held in the ratio of 1:1/2 :1/4 respectively. If these thathus are provoked by any external and internal factors will result as diseases. At this condition these thathoos are called ‘Three Thodam’

Air	+	Space	= Vaatham
Fire			= Piththam
Earth	+	Water	= Kabham

According to the great Saint ‘Yugi muni’ the skin disorders had been classified into 18 types were dealt with in the chapter kuttam. Yugi had not explained Kalanchaga Padai as a separate disease in a detailed manner. Instead he had just made a mention in the classification of 80 vaadha diseases. Such as Kalanchaga vaadham which resembels virpodaga kuttam. The clinical features of Kalanchaga padai more or less correlated with ‘Psoriasis’ as described in modern dermatology.

Inspite of lack literature of this skin diseases it can be clearly stated that root causes of kalanchaga padai is the aggravation of vaadham

“வாதமலாது மேனி கெடாது” - தேரையர்

Moreover the diseases can be formed due to changes in the mind also. Mind is influenced by various stresses in our day to day life on today world. But Siddhars also quoted very anciently that many of the diseases were caused by psychosomatic problems. So that they had advised to control one mind to get ride of stress. This was quoted by Agasthiyar as follows.

“மனமது செம்மையானால் மந்திரஞ் செபிக்க வேண்டாம்  
மனமது செம்மையானால் வாயுவை உயர்த்த வேண்டாம்  
மனமது செம்மையானால் வாசியை நிறுத்த வேண்டாம்  
மனமது செம்மையானால் மந்திரஞ் செம்மையாமே”

- அகத்தியர் குணவாகடம்

From the authors of this diseases study itself the very much thinking of this diseases and collected from the siddha literature which are following.

1. Internaldrug : Shenkottai Nei  
-Siddha Veidhiya thirattu
2. External drug : Sivappu Ennai  
- Siddha Maruthuvam Sirappu

The following pages outline the research work done on kalanchaga padai and the suggestion and recommendation offered for over coming it.

## AIM AND OBJECTIVE

Kalanchaga Padai is by and large an incapacitating disease, bogging down the normal life of a person affected by it. Patient is very much agitated and subjected to a great matter of mentally disturbed and physically suffered. More over as per the expert experiences in medicinal therapy in all walks of medical systems, text books reveals that there would be a permanent cure as well on remedy so far reported. Available therapies are came to study with only remission and reexacerbations of this skin disorder. So for such an intractable disease the world craves for a cure which is the reason why the author has chosen this disease for her dissertation work.

Reason for selecting a shenkottai Nei is that it has equal efficacy to mercury.

The main objective of the present study is to create an awareness about the siddha sciences and to high light the efficacy of sidha drugs among the public.

- ❖ With this basic intention in mind flowing specific objectives have been drawn
- ❖ To thorough references in various siddha literature and modern dermatological books as literature evidences regarding the diseases Kalanchaga Padai.
- ❖ To know the extent of correlation of aetiology, classification, symptomatology, diagnostic methods and the line of treatment on part with modern concepts of dermatology.
- ❖ To conduct a thorough clinical study on Kalanchga padai with internal drug, that is 'Shenkottai Nei' in which Shenkottai is made as a main ingredient. And external drug "Sivappu ennai' in which pungam milk is a main ingredient which are widely indicated in siddha text books are effectively valubale towards skin disorders. There by the author had anxious to go on this trial drug for Kalanchaga Padai.
- ❖ To estimate the efficacy of Shenkottai Nei and Sivappu Ennai.
- ❖ To make a clinical trial with necessary investigations and record all those things with follow – up study of the Patients of Kalanchaga Padai.
- ❖ To have an idea of incidence of Kalanchaga Padai with reference to age, sex, family history, routine life style, habits and season (Paruvakaalam)

- ❖ To have complete study of the disease Kalanchaga Padai, under the headings of
  - a. Pori pulangal
  - b. Udal Kattugal
  - c. Enn vagai thervugal
  - d. Mukkutram etc.

In order to evaluate pathology of this dreadful disease.

- To evaluate the pharmacological study on the trial drug.
- To evaluate the toxicological study on the trial drug.
- To study the bio – chemical analysis on the trial drug.
- To highlight the factors like diet, land, climatic condition and personal hygienic measures in the incidence of Kalanchaga Padai
- For this advise to the patient or by given counselling to the patients about their pshycological factors.
- To make an awarness among the patient about the further occurrence of the same.

## **SIDDHA ASPECTS**

In the Siddha Medicine the Panja boothas (five elements) are elaborately described. Skin is a part of Prithivi (earth). Skin is the largest organ of the human body. It has got a lot of functions each of which is important for the normal physiological functioning of the human body. Some of them are body temperature regulation, excretion, protection from pathogenesis etc.

Man is very much influenced by environment. This principle was much appreciated by the Siddhars which promoted them to say.

‘Microcosams reflects macrocosams’

“அண்டத்திலுள்ளதே பிண்டம்”

Since skin serves as a link between man and universe it is the first organ to be influenced by the change in it. Also since it is the slightest change in mind. Among the panja boothas, Theyu is represented for sleep, appetite, thirst, fearness and unification. If any affection in Theyu, skin is also affected. Any disease affecting the skin causes a socio – economic problem, mental strain and social stigma to the patient.

According to the Siddha Text Book “ Siddha Maruthuvanga Surukkam” skin is divided into six types. They are,

1. Skin containing water
2. Skin having blood
3. Skin which produces scabies
4. Skin which produces kuttam
5. Skin Producing tumour
6. Skin which produces severe pain during an injury

Siddhars embodiments of compassion on living things were naturally moved to write about the aetiology. Clinical features of the diseases and its treatment for this particular debilitating disease called Kalanchaga Padai or Parusethil Noi. And when going for collection of literature for this diseases the author presented with the following details.

### **இயல்(Definition)**

Kalanchaga Padai is a non infectious, inflammatory disease of the skin characterized by well defined erythematous plaques with large adhered silvery (ivory) mica like scales.

நோய் வரும் வழி (Aetiology):

Among the available siddha literature only “Yugi Muni Veithiya Chinthamani” and “Thirumoolar Vaithiyam” are the sources or information on aetiology and clinical features of the 18 types of the skin disorders. There are no specific mention about the specific factors causing Kalanchaga Padai.

“Thirumoolar” has pointed out

“பயில் மொழியீர் திரேகத்தில் கிருமிதானே  
பரந்து திரிகுட்டம் போல் புள்ளி காணும்  
மயலதுவும் கிருமியுந்தா நடந்து புக்கில்  
மேனியது சரசரென வெடித்து புண்ணாகும்” - குருநாடி  
கிருமியால் வந்ததோடம் பெருகவுண்டு  
கேட்க லதின் பிரிவதனைக் கிரமமாக  
பொருமி வரும் வாயுவதும் கிருமியாலே  
திரேகத்தில் சொரி குட்டம் கிருமியாலே  
பெருகிவரும் பவுத்திரமும் கிருமியாலே  
புழுக்கடி போல் காணுமது கிருமியாலே”  
- குருநாடி

The text book of ‘Sirappu – Maruthuvam’ describes the following aetiological factors for Kalanchaga Padai.

1. பரம்பரை நோயில் மூவரில் - Idiopathic and may be genetic  
ஒருவருக்கும்
2. லசன தாபிதம் - Tonsilitis
3. புப்புசப் பிணிகள் - Respiratory Diseases
4. ஒவ்வாமை - Allergic disorder
5. மனஉளைச்சல் - Stress and strain
6. அதிர்ச்சி - Anxiety
7. கால மாறுபாடுகள் - Seasonal variations

Due to Drugs such as

1. தாம்பிரச் செந்துரம் - Red oxide of Copper



2. இளம்பிள்ளை தடுப்பு மருந்து - Polio vaccine
3. குளோரோ குயின் - Chloroquine

முதலிய மருந்துகள் குருக்களையும், படையையும் அதிகரிக்கச் செய்யலாம் என்றும் கூறப்பட்டுள்ளது.

In siddha system of medicine chronic skin diseases are brought under the clinical entity of kuttam.

“Yugi Chinthamani – 800’ describes 18 types of Kuttam (Skin disorders)

‘Thirumoolar’ quotes as follows.

“குட்டமதுவிட கரப்பான் விடநீர் சூலை

சுரோணித்தத்தால் தாதுகெட்டுத் தடிப்புண்டாகும்  
மட்டமறமே கிருமி சென்று மருவும் போது  
வகையாய்க் கிருமியிட விட நீர் சென்று

“குட்டமுடன் தேகமெல்லாம் பறக்கும் போது

குழிகுழியாய்க் கிருமியினீர்க் கொள்ளும் புள்ளி  
தட்டறவே கிருமியிட நீராய் வந்த  
சகல குட்டம் விட கரப்பான் சாற்றலாமே.”

“வியாதியுண் மூவாறு விளங்கிய குட்டங்கேள்

சுயாதி கிரந்தி சுழன் மேகத்தால் ஆறும்  
பயாதி மண்ணுளப் பல வண்டினால் எட்டும்  
நியாதி புழநாலாய் நின்றதிக் குட்டமே.”

Kuttam is common word of chronic skin lesion of various origin.

The Thirumoolar quotes as follows,

“கிரந்தி சுழன்மேகத்தால் ஆறும் பயாதி மண் உளப்பல வண்டினால் எட்டும்,  
நியாதி புழநாலாய் நின்றதிக் குட்டமே”

1. Six types are caused by venereal origin
2. Eight types are caused by insect bite
3. Four types are caused by worms infestation.

‘Agasthiyar’ has mentioned that Kanmam is the main cause for Kutta Noi.

### ***In Agasthiyar Paripoornam – 400***

#### **Kanmavaralauru (Psycho Social Cause):**

“பழவினையால் விஷப் பூச்சி கடித்த தோஷம்  
பாதகர்க்கு ஒரு நாளும் தீர்வதில்லை  
உள்வினையாலாடாடிக் கொள்ள வந்த  
உண்மையது அறியாமல் மூர்க்கஞ் செய்வார்  
களவினையுந் தீர்வதில்லை கடினமெத்த  
கருணையுள்ள பூரணத்தின் கண்காட்சி  
அடவினை நீ காணுமுன்னே அகலச் சொல்லி  
அடையாளம் விரல் குறுகு மின்னங் கேளே.”

- அகத்தியர் பரிபூரணம் 400 - செய்யுள் 214

“விரல் குறுகுங்கால் திமிரும் விஷம் போலேறும்  
மெய்யழுந்துந் தலை சுழலும் வெளுக்குமேனி  
பாரமான தேகமெல்லாந் தடித்துக் காணும்  
பாதமெல்லாம் வெடித்துமிகப் புண்ணாங் காணும்  
சரசமுடன் சொரி கரப்பான் பணம் போல் தோணும்  
சாந்தையாமே விந்துகெடுந் தடித்துவீங்கும்  
நரகுலகி லிந்நோய்க்கு மருந்தீயாதே  
நல்லோரைப் பழித்த குட்டங் கன்மமாமே.”

- அகத்தியர் பரிபூரணம் 400 - செய்யுள் 215

பழவினையால் வரும் நோய்கள் தீர்வதில்லை எனவும், கன்மம் தீரவேண்டும் என்றும் அகத்தியர் பரிபூரணத்தில் கூறப்பட்டுள்ளது.

### **In Agasthiyar Ganma Kandam**

“சேர்ந்த குட்டமொடு குறைநோய்யன் வந்த  
சேதிகேள் மலராத வரும்பு கொய்தல்  
தாரிந்த சீர் செந்து வகைகள் செய்தல்  
தாய்தந்தை மனது நொந்து ரோகந்தானே”

”தானென்ற தெய்வருத் தனைழித்தல்  
சார்வான பெரியோர்கள் தமைப் பழித்தல்  
கானென்ற நந்தவனம் பூஞ்செடிகள் வெட்டல்  
கருமமடா சரீரத்திற் காச போலே  
யூனென்ற வடம்பெல்லாம் மொட்டு மொட்டா  
யுடன் வெளுத்து குறையோயுதிரஞ் சிந்தும்  
வானென்ற கருமங்கள் தீர்ப்பதற்கு  
வரையொன்று சொல்வேன் கேள் நந்தவன்மையே”.

*According to Yugi Vaidhya Chinthamani* classify 18 types of Kuttam in Siddha medicine.

“முத்தான குட்டந்தானே பதினெட்டுக்கும்  
முனியான யூகி நான் சொல்லக் கேளாய்  
பத்தாகும் புண்டரிகக் குட்டத்தோடு  
போருகின்ற விற்போடக குட்டமாகும்  
பத்தாகும் பாமக்குட்டம், கசசர்மகுட்டம்  
பரிவான கர்ணகுட்டம், சிகுரகுட்டம்  
கித்தாகும் கிருட்டிணக் குட்டம், அவுதாம்பரக்குட்டம்  
கெடியான மண்டலகுட்டமுமா மென்னே  
குட்டமாம் பாப்பரிசு குட்டமோடு  
குடிமாம் விசார்ச்சிகக் குட்டமாம்  
வட்டமாம் வையாதி குட்டமோடு  
மருவலா கீடப குட்ட சர்மதல  
திட்டமாந் தேத்திரு குட்டமோடு  
சித்துமா, குட்டம், சதாரு குட்டம்  
துட்டமாரு குட்டந், தன்னொடொக்க  
சுயம்பான பதினெட்டு குட்டமாச்சே.”

- |                |                  |                  |              |
|----------------|------------------|------------------|--------------|
| 1. Pundareegam | - Padar thamarai | 10 Abarisam      | - Vali       |
| 2. Virpodagam  | - Koppulam       | 11. Visharchigam | - Sori       |
| 3. Bamam       | - Sirangu        | 12. Vibadhigam   | - Senkuttam  |
| 4. Gaja Sarmam | - Yaanai thol    | 13. Sarmathalam  | - Tholvedi   |
| 5. Karnam      | - Kaadhu         | 14. Kideepam     | - Pantrithol |
| 6. Sikuram     | - Tholperunoi    | 15. Theththuru   | - Thaddipu   |

7. Krishnam	- Karuperunoi	16. Sithma	- Naa
8. Avudhumbaram	- Athikkai	17. Sadharu	- Purai
9. Mandalam	- Valayam	18. Suwedham	- Venkuttam

விர்போடகம், பாமம், கஜசர்மம், கிருட்டிணம், அவதூம்பரம்,  
தேத்துரு, சித்துமா, கிடிபம், சதாரு, சருமம் ஆகிய குட்டங்கள் தீரும்.

In Yugimuni 800, Yugi mentioned the following causes for kuttam

1. Excessive intake of fish, snail, crab
2. Doing Yoga practice immediately after intake of diet
3. Excessive chillness, excessive hot, excessive sleeping, mental stress.
4. Routing diet sometimes combines along with unwanted things like sand, hair etc.

In Yugimuni 800,

“ஆச்சென்ற பதினெட்டு குட்டந்தானும்  
அவரவர்கள் செய்கின்ற அதர்மத்தாலாம்  
தேச்சென்ற சிவாலயத்திலுச் சிட்டங்கள்  
செய்தவர்கள் சிவ நிந்தை பண்ணினோர்கள்  
மூச்சென்ற பெரியோரைத் தூஷித்தோர்கள்  
மூர்க்கமாய் அடைக்கலத்தை எடுக்கின்றோர்கள்  
கூச்சென்ற திணையளவின் குறைந்த கூலி  
கொடுக்கின்றோர் குட்டத்திற் கூடுவாரே”

- யுகி முனி 800 - பாடல 496

Yugi Muni described only psycho – socio factors as the main cause. They are stress inducing factors, he had attributed the following of causes.

1. Misbehaviour in the temple
2. Sacrilege towards god
3. Abusing the geriatric people
4. Breach of trust
5. Paying low wages to the workers.

The main factors behind reasons one manifestation of stress which can be considered as precipitating factor. The psycho tranquility of the individual depends upon the harming of social movements. Any adjustment disorder will affect the well being of the individual as well as society.

In Yugi Chinthamani among the eighteen types of skin (kuttam) diseases, the clinical features of three types resemble as Kalanchaga Padai. But no description is available in Siddha literature under the heading of Kalanchaga Padai.

The three types of Kuttam are:

1. Thethuru Kuttam
2. Sadharu Kuttam and
3. Virpodaga Kuttam

### **1. Thethuru Kuttam**

#### **Clinical Features:**

“சர்மந்தான் சிவப்பாக வட்டதணிதுச்  
சலவைபோல் வெளுக்குமே தினவுண்டாகும்  
கர்மந்தான் ரோகமது மிகவுண்டாகும்  
மயிரெல்லாஞ் சுரண்டுமே உருண்டையாகும்  
கர்மந்தான் பித்த சேட்டும மிகுக்கும்  
காயந்தான் கதித்துமே திமிருண்டாகும்  
தர்மந்தான் சடமெல்லா மூதலாகும்  
தாக்கான தேத்திருக் குட்டந்தானே”

- யுகி முனி வைத்திய சிந்தாமணி 800 பாடல், 511

Under “Thethuru Kuttam”, annular erythematous lesions with the white appearance, itching, oedema of the body and rolling of hairs like balls are the characteristic clinical features in this entity.

### **2. Sadharu Kuttam**

#### **Clinical Features**

“சித்தான தண்டிப்பாய் ரத்தவர்ணம்  
செழும்பச்சை வெள்ளையாய்ச் சிவப்புமாகும்  
எத்தான வெரிப்போடு தினவுமாகும்

எளிதான சேட்டுமவாதத் துற்பத்தி  
பத்தான கரடிகட்டிப் புண்ணுமாகும்  
பாம்பு தோல் போற்றிரைந்து பருத்துக் காணும்  
வித்தான முக்கோடு காது கன்னம்  
மிகத்துடிப்பாக் சதாரு குஷ்டந்தானே”

Erythematous patches, Burning, Itching, patches covered with white silvery scales thickening of the eyes, cheek & nose are the clinical features of satharu kuttam.

### 3. Virpodaga Kuttam

Clinical features:

“புதுமையாய்ச் சரீரமெங்குந் தினவுண்டாகும்  
பொருவெடியாய்த் திக்கெனத்தீக் கொழுந்து போல்  
மெதுமையாய் விட்டெரியும் நல்ல பாம்பின்  
விஷப்படம் போல தடித்து வெளுப்புமாகும்  
கதுமையாய் மிகச் சொரிய சிவப்புமாகும்  
தூக்கமொடு சஞ்சலமும் மிகவுண்டாகும்  
கதுமையாய் தோலெல்லாந் தடிப்புண்டாகும்  
கனத்தவிற் போடகமான குட்டந்தானே”

- யுகி முனி பெருநூல் 800, பாடல் - 498

A skin erythematous lesions with swelling white appearance and itching are described. Usually these entities are associated with anxiety and despair.

In the text book ‘Siddha Maruthuvam Sirappu’ author by Dr. R. Thiagarajan, LIM describes as,

1. The skin lesions are red in colour and raised margin the lesions have white silvery and rough thick scales with pin point bleeding.
2. There are variation in the size and shape
3. In children these lesion may be like water drops and these may occur in scalp and in face. Sometimes as per the severity it seems all over the body.

### Psoriatic Arthropathy (காளாஞ்சக வாதம்)

Kalanchaga Padai is often associated with painful arthritis known as Kalanchaga Vaatham. Cases in chronic stage some arthritis incapacitates to resort to hospitalization. The most often affected joints are interphalangeal joints.

The terminal interphalangeal joints are usually involved as opposed to the proximal interphalangeal joints in “Azhai keel Vaayu” which is identical with Rheumatoid Arthritis. In these cases the affected fingers show nail changes. This combination is termed “Psoriatic arthropathica”.

The joints of fingers, ankles, knee and sacroiliac are selectively affected. Those joints are swollen and painful with psoriatic lesions. Radiological changes show gross changes in the affected area. These are characteristic and consist of osteoporosis followed by decreased density, diminished joint space, erosion of joint surfaces followed by eventual destruction of the end bones.

Yugimuni describes the clinical features of Kalanchaga Vaatham as follows.

மாத மாங் கால் கையில் குரங்கிரண்டும்  
வருந்து சந்து முறுக்கியே குடைந்து நொந்து  
நாதமா நடைதானுந் தான் கொடாமல்  
நலிந்துமே முடமாகிக் கரடு கட்டிச்  
சேதமாஞ் சடந்தானு மிக வெளுத்துத்  
தினவோடு சிரங்குமாய்ச் சேட்பமாகிக்  
காதமாய் ருசியொடு மயக்கமாகும்  
கருதிய காளாஞ் சகமாம் வாதமாமே.  
- யுகி வைத்திய சிந்தாமணி செய்யுள் 259.

Pain in the major joints with immobility, pustules, itching, anorexia, fatigue etc, are the clinical features of Kalanchaga Vaatham

In Chronic Cases

1. The skin lesion occurs over fore arms skin.
2. In Some, these patches appear over the palm and soles.
3. In some, the patches occur all over the body.
4. The patches are coin shaped over them. The shape may be either round or oval.
5. In obese women the lesion may occur over inguinal region, axilla with muscle foldings with wet.

6. One fourth of patches have lesion over nails pitting in nature.
7. 7% of patients develop affection of joints as psoriatic arthropathy.

### **நோய் கணிப்பு (Diagnosis):**

Piniyari Muraimai is a method of diagnosing a disease (affecting the mankind) it is based upon three main principles;

1. Poriylarithal (Inspection)
2. Pulanalarithal (Palpation)
3. Vinathal (Interrogation)

Physicians ‘Pori’ and Pulan’ are used as tools for examining the ‘pori pulan’ of the patient. The above principles correspond to the methodology of 1. Inspection, 2. Interrogation and 3. Palpation in modern medicine, for arriving a clinical diagnosis of the disease.

### **1. Poriylarithal (Inspection)**

Pori is considered as the five senses of perception namely

1. Nose
2. Tongue
3. Eye
4. Skin
5. Ear

‘Poriylarithal’ is examining the pori of the patient by the physician for diagnosing.

### **2. Pulanalarithal (Palpation)**

‘Pulan’ are five object of senses. They are,

1. Smell
2. Taste
3. Vision
4. Sensation to touch
5. Hearing

‘Pulanalarithal’ means examination of the ‘pulan’ of the patient by the physician for diagnosing purpose.

### **3. Vinathal (Interrogation)**



Vinathal is gathering the informations regarding the history of diseases, its clinical features etc, from the patient or his close relatives who are taking care of him, when the patient is not in a position to speak or of the patient is a child.

### அளவைகள் (Logics)

Alavaigal are used in clinical diagnose of a disease.

அளவை காண்டல் கருதல் உரை அபாவம் பொருள் ஒப்பாற்றென்பர்  
அளவை மேலும் ஒமிபுண்மை யைதிகத் தோடியல் பென நான்  
களவை காண்பர் அவையிற்றின் மேலும் அறைவர் அவையெல்லாம்  
அளவை காண்டல் கருதல், உரை என்றும் மூன்றிலடங்கிடுமே.

- சிவசித்தியார் அளவை எண். 6

Alavai is divided in to ten types, they are

1.	Observation	-	காண்டல்	6.	Comparison	-	உபமானம்
2.	Inference	-	கருதல்	7.	Inference by elimination	-	பாரிசேஷம்
3.	Authority, Literature	-	ஊரை	8.	Probability	-	சம்பவம்
4.	Preception	-	அபாவம்	9.	Tradition	-	ஐதீகம்
5.	Presumption	-	அருத்தப்பத்தி	10.	Natural Inference	-	இயல்பு

The above mentioned “ten alavaigal” the main three alavaigal are,

#### 1. Kaandal

Through ‘ kaandal’ the physician can directly see the patien and hear the patients all the complaints and at length concludes a diagnosis.

#### 2. Karuthal

Through Envagai thervu, Neerkuri and Neikuri we can diagnose a disease by Karuthal.

#### 3. Urai (Literature evidence of Siddha)

Comparative study of the signs and symptoms of the patient with the reference books and come to a diagnosis.

## **Ennvagai thervugal (Eight diagnostic tools)**

Siddhars have developed a unique method of diagnosing the disease by “Ennvagai thervugal”

“நாடி ஸ்பரிசம் நா நிறம் மொழி விழி

மலம் மூத்தரமிவை மருத்துவராயுதம்”

- நோய் நாடல் நோய் முதல் நாடல் (முதல் பாகம்)

மெய்க்குறி நிறம் தொனி விழி நா இருமலம் கைக்குறி

- தேரையர்

Hence the diagnosis is made by the following.

- |             |                      |              |         |
|-------------|----------------------|--------------|---------|
| 1. Naadi    | (Pulse)              | 5. Mozhi     | (Voice) |
| 2. Sparisam | (Sensation to Touch) | 6. Vizhi     | (Eyes)  |
| 3. Naa      | (Tongue)             | 7. Malam     | (Feces) |
| 4. Niram    | Colour               | 8. Moothiram | (Urine) |

The specialty of eight tools of diagnosis is mentioned in the following verses also.

## **Kalanjacha Padai in relation with Ennvagai thervugal**

### **1. Naadi (Pulse)**

உடலில் உயிர் தரித்திருப்பதற்குக் காரணமான சக்தி எதுவோ அதுவே தாது அல்லது நாடி எனப்படும்.

“நாடி என்றால் நாடியல்ல, நரம்பில் தானே

நலமாகத் துடிக்கின்ற துடிதானுமல்ல

நாடி என்றால் வாத பித்த சிலேற்பனமுமல்ல

நாடி எழுபத்திராயிரந் தானுமல்ல

நாடி என்றால் அண்ட பேரண்டமெல்லாம்  
நாடி எழுவகைத் தோற்றத்துள்ளாய் நின்ற  
நாடியதுயா ராய்ந்து பார்த்தாரானால்  
நாடியுறும் பொருள் தெரிந்து நாடு வாரே”.

- பதினெண் சித்தர் சதக நாடி நூல்

Naadi is responsible for the existence of life and can be felt one inch proximal to the wrist on the radial side by means of palpation with the tips of index, middle and ring fingers corresponding vaatham, Piththam and kabam respectively.

The three humours vaatham, Piththam and kabam exists in the ratio 1: ½ : ¼ normally. Derangement in these ratios leads to various disease entities.

The three “Uyir thathukal” are formed by the combination of three naadigal with three Vaayu.

- |               |   |          |   |          |
|---------------|---|----------|---|----------|
| a) Edakalai   | + | Abaanan  | = | Vaatham  |
| b) Pinkalai   | + | Piraanan | = | Piththam |
| c) Suzhumunai | + | Samaanan | = | Kabham   |

In kalanchaha padai the following types of naadi can be seen commonly.

They are,

- a) Vaatha Piththam
- b) Piththa vaatham
- c) Kaba Piththam

## II. Sparism

In case of Kalanchaga Padai well defined macules, papules, thickening, roughness, pain and white silvery scaling of the skin can be noticed at the affected area.

## III. Naa

No abnormality is seen in Naa

## IV. Niram

White patches with silvery scales can be noticed at affected areas.

## V. Mozhi

No abnormality was ruled out.

## VI. Vizhi

No abnormality is seen in vizhi.

## VII. Malam

Constipation was reported in some cases.

## VIII. Moothiram

Collection of urine for the determination of Neerkuri and Neikuri, a special diagnostic method.

### Neerkuri and Neikuri

“அருந்து மாறிரதமும் அவிரோமதாய்  
அ.கல அலவர்தல் அகாலவூன் தவிர்ந்தழற்  
குற்றள வருந்தி உறங்கி வைகறை  
ஆடிக் கலசத் தாவியே காதுபெய்  
தோரு முகூர்த்தக் கரைகுட் படுநீரின்  
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”.

- சித்த மருத்துவாங்க சுருக்கம்

Prior to the day of urine examination the patient is instructed to take a balanced diet and quantities of food must be proportionate to his routine in take. The patient could have no disturbed sleep. After waking up in the morning, the first urine voided is collected in a clear wide mouthed glass container and is subjected to analysis of “neerkuri and neikuri” with in one and a half an hour. Then, neerkuri is to be found out by

### Neerkuri

“வந்த நீர்க்கரி யைடை மணம் நுரை எஞ்சலென்  
றைந்திய லுளவை யறைகுது முறையே”

- சித்த மருத்துவாங்க சுருக்கம்

Voided urine has the following characters

- |          |   |                  |
|----------|---|------------------|
| 1. Niram | - | Colouration      |
| 2. Edai  | - | Specific gravity |
| 3. Manam | - | Smell            |
| 4. Nurai | - | Frothy nature    |

##### 5. Enjal - Quantity of urine voided

Apart from these, the frequency of urination, abnormal constituents, such as sugar, protein, presence of blood, pus, renal calculus crystals also be found out.

In Kalanchaga padai patient straw or hay coloured urine is noticed.

##### Neikuri:

The speciality of neikuri is stated in the following verse.

“ஐயக்குறி கொடுவட வானிழ லமர்ந்தோர்  
கைக்குறி தெரித்த நங்கடவுளைத் துதித்தே  
மெய்க்குறி நிறந்தொனி விழிநா இருமலம்  
கைக்குறி முழுவதா உங்கற்றார் தம்மினும்  
பொய்க்குறி மெய்க்குறி புகலு மெவர்க்கும்  
நெய்க்குறி அதனை இந்நீணிலத் துரைப்பாம்”.

- சித்த மருத்துவ நோய் நாடல் நோய் முதனாடல் திரட்டு

##### The Process of dropped gingely oil indication

“நிக்குறிக் குரைத்த நிருமாண நீரிற்  
சிறக்க வெண்ணெய்யோர் சிறுதுளி நடுவிடுத்  
தென்றறத் திறந்தொலி ஏகாதமைத்ததி  
னின்றதிவலை போம் நெறிவிழியறியவும்  
சென்றது புகலுஞ் செய்தியை யுணரே”

- சித்த மருத்துவ நோய் நாடல் நோய் முதனாடல் திரட்டு

The collected specimen as said above is to be analysed by following method. The specimen is kept open in a glass dish or hina clay container. It is to be examined under direct sunlight, without any shaking of the vessel. Then add on drop of gingely oil by at a distance of 1/2” or 3/4” height observe keenly the direction it spreads with in few minutes, and conclude the diagnosis as follows.

“அரவென நீண்டின் அ.:தே வாதம்  
ஆழி போல் பரவின் அ.:தே பித்தம்  
முத்தொத்து நிற்கின் மொழிவதென் கபமே  
அரவிலாழியும் ஆழியில் அரவும்  
அரவின் முத்தும் ஆழியில் முத்தும்”.

- சித்த மருத்துவ நோய் நாடல் நோய் முதனாடல் திரட்டு

##### Paruvakaalm (Seasonal Variation):

Sl.No	Kalam	Kuttram	State of Kuttram	Suvai
1.	Kaar Kaalam (Aavani – Puratasi) (Aug 16 – Oct 15)	Vaatham↑↑ Piththam↑	Vettrunilai Valarchi Thannilai Valarch	Enippu Pulippu Uppu
2.	Koothir kaalam (Iypasi – Karthigai) (Oct 16 – Dec 15)	Vaatham (-) Piththam ↑↑	Thannilai Adaithal Vettrunilai Valarchi	Enippu Kaippu Thuvvarppu
3.	Munpanikaalam (Markazhi – Thai) (Dec 19 – Feb 15)	Piththam (-)	Thannilai Adaithal	Enippu Pulippu Uppu
4.	Pinpanikaalam (Masi – Panguni) (Feb 16 – Apr 15)	Kabam ↑	Thanniklai Valarchi	Enippu Pulippu Thuvvarppu
5.	Elevenil kaalam (Chithirai – Vaikasi) (Apr 16 – Jun 15)	Kabam ↑↑	Vetrunilai Valarchi	Kaippu Karppu Thuvvarppu
6.	Mudhuvenil kaalam (Aani – Aadi) (Jun 16 – Aug 15)	Vaatham ↑ Kabam (-)	Thannilai Valarchi Thannilai Adaithal	Enippu

### Five Types of Lands

It is divided in to five types.

1. Kurinji : Mountain regions and surroundings
2. Mullai : Forest regions and surroundings
3. Marutham : Cultivating regions and surroundings
4. Neithal : Sea and coastal region
5. Palai : Desert land only

### Udal Kattugal

Our body consists of seven udal kattugal. It gives strength and structure to our body.

Sl.No	Udal kattugal	Functions
1.	<b>Saaram</b>	It gives strength to the body and mind
2.	<b>Senneer</b>	Saram after absorption is converted into senneer. It is responsible for knowledge, strength, boldness and healthy complexion.
3.	<b>Oon</b>	Gives structure and shape to the body and is responsible for the movements of the body.
4.	<b>Kozhuppu</b>	Lubricates the organs and proceed on its own works.
5.	<b>Enbu</b>	Protects the vital organs and used for movements and nominates body structure
6.	<b>Moolai</b>	Present inside the bones and it gives strength and maintains the normal condition of the bone.
7.	<b>Sukkilam</b> (or) <b>Suronitham</b>	Responsible for the reproductive function of species.

Sl.No	Udal Kattugal	Increased Conditions	Decreased Conditions
1.	<b>Saaram</b>	Leads to disease identical to the increase in Kabha like loss of appetite, excessive salivation	Loss of weight, tiredness, dryness of skin, laziness, diminished activity of the sense organs
2.	<b>Senneer</b>	Boils and tumours in different parts of the body, splenomegaly Colic pain, increased blood pressure, reddish eye and skin, jaundice, leprosy, haematuria etc.	Tiredness, Lassitude, anaemia
3.	<b>Oon</b>	Tumours or extra growth around	Muscle wasting

		the neck, face, abdomen, thigh, genitalia etc.	
4.	<b>Kozhuppu</b>	Identical to that of increased oon associated with dyspnea and loss of activity	Pain
5.	<b>Enbu</b>	Strong bones and teeth	Weak bones, teeth, nails and hairs
6.	<b>Moolai</b>	Heaviness, swollen eyes, swollen phalanges, oliguria and non – healing ulcers	Ostoporosis and shunken eyes
7.	<b>Sukkilam</b> (or) <b>Suronitham</b>	Increased sexual activity and signs identical to urinary calculi	Failure to reproduce, pain in genitalia etc.

In the case of Kalanchaga Padai out of seven udalkattugal saaram, senneer, oon and enbu commonly affected.

Saaram : Dryness, roughness, tiredness

Senneer : Dryness, paleness of the skin

Oon : Weakness of the sense organs

Enbu : Pain in the knee joints

## Mukkutram

Human body is influenced by Three Thodam (ie) Vaatham, Piththam and Kabam. They are responsible for normal physiological condition of the body.

## Vaatham

Vaatham is a kinetic energy, which influences all movements.

Vaatham is located in abaanan, idakalai, feces, spermatic cord, illiac bone, skin, nerves, joints, hair follicles, muscles, bone, ear and thigh.

Sl.No	Name	Locations	Physiologic functions
1.	Piranan	Heart and Lower and Upper Respiratory Tracts	Controls knowledge, mind and five objects of sense useful for breathing



2.	Abanan	Lower abdomen and extremities	Responsible for urination, expels faeces and foetus, discharge sperm and menstruation.
3.	Viyanan	Mainly at heart	Responsible for movement of all parts of the body and used to feel the sensation
4.	Uthanan	Chest	Responsible for vomiting cough, hiccough, sneezing
5.	Samanan	Stomach	Aids for proper digestion. It controls the activity of other Vaayus
6.	Naagan	Eyes	Responsible for opening and closing of the eyes
7.	Koorman	Heart and Eyes	Responsible for vision and yawning and controls lacrimation
8.	Kirukaran	Throat	Responsible for salivation nasal secretion and appetite
9.	TheVaathathan	Eruvaai & Karuvaai	For laziness, sleeping and anger
10.	Thananjeyan	Nose	Responsible for bloating of the body after death. It escapes on the third day after death through the cranium when it bursts.

In the case of Kalanchaga Padai

1. Abaanan - Habitual constipation
2. Viyaanan - Erythematous in the affected lesions of skin
3. DeVaathathan - Insomnia like condition

The above Vaayus are affected commonly

### **Piththam**

Piththam is responsible for all the transformation. Piththam is located in urinary bladder, heart, head, umbilicus, abdomen, blood, sweat, skin and eye.

Piththam is classified into 5 types. They are,

1. Anala Piththam - Responsible for digestion of food

- |                     |   |  |
|---------------------|---|--|
| 2. Ranjaga Piththam | - | Responsible for colour of blood  |
| 3. Saathagam        | - | Located in heart and is responsible for normal activities of the body. |
| 4. Aalosagam        | - | Responsible for normal vision  |
| 5. Praasagam        | - | Responsible for the complexion of skin.                                |

In case of Kalanchaga padai

- |                   |   |  |
|-------------------|---|--|
| 1. Anala Piththam | - | Indigestion of food  |
| 2. Ranjagam       | - | Paleness of the conjunctiva and tongue                     |
| 3. Saathagam      | - | Difficulty to do the routine works properly & sluggishness |
| 4. Praasagam      | - | Dryness and roughness of skin                              |

### **Kabam**

Stabilizes, maintains and lubricates all movements.

Kabam is found in samaanan, semen, brain, head, tongue, nose, bones, bone marrow, fat, nerves, chest, blood, large intestine, eye, stomach and pancreas.

Kabam is classified in to 5 types, they are

- |                |   |  |
|----------------|---|--|
| 1. Avalambagam | : | Heart is the center for avalambagam. It controls all other forms of kabam  |
| 2. Kilethagam  | : | Stomach is the center for kilethagam. It give moisture and softness to the ingested food and helps for digestion |
| 3. Pothagam    | : | Tongue is the center for pothagam and it is responsible for the sense of taste                                   |
| 4. Dharpagam   | : | Head is the center for dharpagam. It gives cooling effect to eyes  |

5. Santhigam : It lies in the joints and is responsible for the locomotive action of movable bony joints.

In case of kalanchaga padai,

- Santhigam : Pain in knee joints & elbow and interphalangeal joints are affected.

### **Abnormal functions of Vaatham**

Pain in the wholebody, twitching, piercing pain, inflammation, redness of the complexion also roughness of the skin. Hardness of the limbs, astringent taste, sweating, sleep contraction and numbness or paralysis of the limb, tremors, muscular wasting, severe pain, decrease in the amount of excretion of stools and urine, thirst, blackish discolouration of the skin, stools, urine and muddy conjunctiva.

### **Abnormal Functions of Kabham**

Pale skin complexion, cold, itching, dullness, heaviness, oiliness, loss of sensation, a sense of sweetness in mouth.

Humor	Increase	Decrease
Vaatham	Distended abdomen, Constipation, Weakness, Insomnia, Tremors Breathlessness, Blackish discoloration	Body pain, Feeble Voice, Syncope, Diminished capability of brain
Piththam	Yellowish discoloration of	Cold,

	eyes, skin, urine and motion Polyphagia, Polydypsia, Burning sensation all over the body, Sleeplessness	Pallor, Decreased appetite Symptoms associated with growth of kabam
Kabam	Loss of appetite, Excessive salivation, Heaviness, Dyspnoea, Excessive sleeping, Whiteness, Diminished activity	Prominence of bone edges, Dry cough, Lightness, Profuse sweating, Palpitation, Giddiness, Dryness of joints

#### Relation between Suvai, Panjabootha and Mukkutram

Sl.No	Suvai	Panjabootha	Mukkutram
1.	Enippu (Sweet)	Piruthivi + Appu	Kabha ↑ Vaatha ↓ (-) Piththa ↓ (-)
2.	Pulippu (Sour)	Piruthivi + Theyu	Kabha ↑ Piththa ↑ Vaatha ↓ (-)
3.	Uppu (Salty)	Appu + Theyu	Kabha ↑ Piththa ↑ Vaatha ↓ (-)
4.	Kaippu (Bitter)	Vaayu + Space	Vaatha ↑ Kabha ↓ (-)

			Piththa ↓ (-)
5.	Karppu (Pungent)	Vaayu + Theyu	Vaatha ↑ Piththa ↑ Kabha ↓ (-)
6.	Thuvarppu (Astringent)	Piruthivi + Vaayu	Vaatha ↑ Kabha ↓ (-) Piththa ↓ (-)

↑ - Valarchi

↓ - Samappaduthuthal

### Udal Vanmai – Body Immunity

The Udal Vanmai is classified in to 3 types. They are,

1. Iyarkkai Vanmai
2. Seyarkkai Vanmai
3. Kaala Vanmai

#### 1. Iyarkkai Vanmai :

Natural immunity of the body itself by birth.

#### 2. Seyarkkai Vanmai:

Improving the health by intake of nutritious food materials, activities and medicines.

#### 3. Kaala Vanmai:

Development of immunity according to age and environment

When Udal vanmai is affected there may be a possibility affection in Kalanchaga padai.

### Gnanenthiriyam.

Gnanenthiriyam are Mei, Vaai, Kan, Mookku and Sevi

1. Mei : Feels all types of sensation
2. Vaai : For recognize taste
3. Kann : Meant for vision
4. Mookku : For recognize smell

**5. Sevi** : For hearing

In case of ‘Kalanchaga Padai’.

**1. Mei** : Roughness of the skin, white silvery scales are affected generally. Other are not affected

### **Kanmenthriyam**

Kanmenthriyam are kai, kaal, vaai, eruvaai and karuvaai

- 1. Kai** : Majority of normal works done by hands
- 2. Kaal** : For Walking
- 3. Vaai** : For Speaking
- 4. Eruvaai** : For defaecation
- 5. Karuvaai** : For reproduction

In case of ‘Kalanchaga padai’

**1. Kai, Kaal** : Difficult to use the limbs in this stage of kalanchaga Vaatham

### **Line of Treatment**

In Siddha system, the main aim of the treatment is cure Udarpini (due to Mukkuttram) and Manappini (due to changes in Mukkunam). Treatment is not only for perfect healing but also for the prevention and rejuvenation.

Thiruvalluvar says about physicians duty, study the disease, study the cause, seek subsiding ways and do what is proper and effective.

“நோய் நாடி நோய் முதல் நாடி அது தணிக்கும்  
வாய் நாடி வாய்ப்பச் செயல்”.

“உற்றானளவும் பிணியளவுங் காலமும்  
கற்றான் கருதிச் செயல்”

- திருக்குறள்

So it is essential to know the disease, the aetiology, the nature of the patient, severity of the illness, the seasons and the time of occurrence must be observed clearly.

Line of treatment is as follows:

Kaappu (prevention)

Neekkam (Treatment)

Niraivu (Retoration)

### **Kaappu: (Prevention)**

As per Siddha system even during the time of conception the vinaipayan is transferred into the fertilized embryo, which is aetiology for certain diseases may be cured not only by medicines but by teaching the following habits.

1. Teaching good moral habits.
2. Avoid Stress and strain.
3. Taking purgatives once in 6 months.
4. Always have good mental thoughts by doing meditation.
5. Yoga

### **Yoga:**

Skin is the reflex of mind and so we should treat not only the physical but also treat mind and soul. Thereby patients were advised to do yoga practice i.e. Pranayamam, Aasanas like Pathmaasana Sarvaangasana and Poorna savasanthi aasanam. These aasanas relieve patient's stress and strain and also useful in kalanchagapadai disease.

### **பத்மாசனம்**

சமதளத்தில் சம்மணமிட்டு உட்கார்ந்து வலப்பாதத்தை இடத் தொடை மீதும், இடப்பாதத்தை வலது தொடை மீதும் ஏற்றி இரண்டு கைகளையும் முன்புளரம் ஒன்றன் மீது ஒன்றாக மலர்ந்திருக்குமாறு இருத்தல். இதனால் உடல் நலமும் மன மகிழ்ச்சியும் ஏற்படும்.

### **சர்வாங்காசனம்**

மல்லாந்து படுத்து கால்களை மெதுவாக ஓட்டியபடியே மேலே தூக்கிப் பின் புட்ட பாகத்தையும் இடுப்புப் பாகத்தையும் மேலே தூக்கிக் கைகளால் முதுகுப் புறத்தில் தாங்கி நிற்கல்.

வீதன கோளம் தூண்டப்படுகிறது (Chin with sternum)

நரை, திரை, மூப்பு மாறி இளமையுண்டாகும்

பெருநோய் தீரும்

உடலின் எல்லா உறுப்புகளும் பலப்படும்.

### பூரண சுவசாந்தி ஆசனம்

மல்லாந்து படுத்துக் கால்களை நேராயும் கைகளை உடலோடு பக்கவாட்டிலும் வைத்து நேராகப் படுத்து இருத்தல்

இது களைப்பைப் போக்கும் புத்துணர்ச்சியை உண்டாக்கும்

All the patients were also avised to follow Siddhars preventive measures which would give immortality of body and soul, quoted in *Pathartha Guna Chinthamani* as follows.

“திண்ண மிரண்டுள்ளெ சிக்க வடக்காமந்

பெண்ணின்பா லொன்றைப் பெருக்காமல் - உண்ணுங்கால்

நீசருக்கி மோர் பெருக்கி, நெய்யுருக்கி யுண்பவர்தம்

பேருரைக்கிற் போமே பிணி”

“பாலுண்போம்; எண்ணெய்பெறின் வெந்நீரிற் குளிப்போம் ;

பகற்புணரோம்; பகற்றுயிலோம் ; பயோதரமு மூத்த

ஏலஞ்சேர் குழலியரோ டிளவெயிலும் விருப்போம்

இரண்டடக்கோம் ; ஒன்றைவிடோம் ; இடது கையிற் படுப்போம்

மூலஞ்சேர் கறி நுகரோம் மூத்த தயிர் உண்போம் ;

முதனாளிற் சமைத்தகறி யமுதெனினு மருந்தோம் ;

ஞாலந்தான் வந்திடினும் பசித்தொழிய வுண்ணோம்

நமனாக்கிங் கேதுகவை நாமிருக்கு மிடத்தே.”

“உண்பதிரு பொழுதொழிய மூன்று பொழுதுண்ணோம் ;

உறங்குவதி ராவொழியப் பகலுறக்கஞ் செய்வோம் ;

பெண் கடமைத் திங்களுக்கோர் காலன்றி மருவோம் ;

பெருந்தாக மெடுத்திடினும் பெயர்த்து - நீரருந்தோம்”

மண் பரவு கிழங்குகளிற் கருணையின்றி புசியோம்;



வாழையிளம் பிஞ்சொழியக் கனியந்தல் செய்வோம் ;  
நண்புபெற வுண்டபின்பு குறநடையுங் கொள்வோம்  
நமனார்க்கிங் கேதுகவை நாமிருக்குமிடத்தே”.

“ஆறு திங்கட் கொரு தடவை வமன மருந் தயில்வோம்  
அடர் நான்கு மதிக்கொருகாற் பேதியுறை நுகர்வோம்  
தேறுமதி யொன்றரைக்கோர் தரநசியம் பெறுவோம்  
திங்களரைக் கிரண்டுதரஞ் சவளவிருப்புறுவோம்  
வீறுசதுர் நாட்கொருகால் நெய்கொருகா லிடுவோம்  
நாறுகந்தம் புட்மலை நடுநிசியின் முகரோம்  
நமனார்க்கிங் கேதுகவை நாமிருக்கு மிடத்தே

“பகத்தொழுக்கு மாதரசங் கரந்துடைப்ப மிவை ந்தூட்  
படநெருங்கோம் ; தீப மைந்தர் மரநிழலில் வசியோம் ;  
சுகப்புணர்ச்சி யசனபக னத்தருணஞ் செய்யோம் ;  
துஞ்சலுண விருமலஞ்சை யோகமழுக் காடை  
வகுப்பெடுக்கிற் சிந்துகச மிவை மாலை விருப்போம்  
வற்சலந்தெய் வம்பிதுர்சற் குரவை விட மாட்டோம்  
நகச்சலமு முளைச்சலமுந் தெறிக்குமிட மணுகோம்  
நமனார்க்கிங் கேதுகவை நாமிருக்கு மிடத்தே”

- பதார்த்த குண சிந்தாமணி

## Neekkam (Treatment)

The aim of treatment is based on

a) To bring the three thodams in to normal equilibrium state by purgation with.

“Poovarasam pattai ennai”

b) To treat the patient according to symptoms, by internal medicine

“Shenkottai Nei” as well as external medicine Sivappu ennai.

For normalizing three thodams,

“விரசனத்தால் வாதம் தாமும்

வமனத்தால் பித்தம் தாமும்

நசிய அஞ்சனத்தால் கபம் தாமும்.”

Here Kalanchaga padai, Vaatha humor is deranged mainly. Administration of laxatives or purgatives to the patient brings the vitiate Vaatha into normal. The patients were derived to use “Nalunguma” instead of soap.

The patients were also advised to wear clean cotton clothes and to avoid cosmetics for allergic reactions.

### **Restriction Regarding Food Habits**

1. Avoid Karappan food items.

“பெருகுஞ் சோள மிறுகும் பெரும் கம்பு  
வரகு காருடன் வாழையின் காயொடு  
உரைகொள் பாகற் கெளிற்று மீன் உண்டிடல்  
விரிவதாய்க் கரப்பானு மிகுந்ததே”

- பதார்த்த குண சிந்தாமணி

2. Avoid bitter Millet, Maize, Unripened banana, Bitter guard, Pumpkin, Tubers, Banana, Citrus Fruits, Brinjal, Tomato, Ladies Finger, Tamarind, Spices.
3. Avoid chicken, Fish, Dry fish, Egg, Artificial Food colours.
4. Obese must be restricted.
5. Avoid Alcohol, Cigarette / Beedi, Betel nut, Tobacco.
6. Avoid Narcotic drugs, Medications like penicillin etc, Chemicals, paints, fertilizers, cold air, occupational allergens.
7. Should not be stressed and strained.
8. Adapt regular exercise keep regular bowel movement.
9. Since it is chronic and not a life threatening disease, it should not be loaded with heavy drugs.
10. The medications and meditations should calm the mind just free from stress and strain.
11. The patches should be washed with lukewarm water to remove the scales every day early morning and after the bath when moisture of the lesions gets dried, the external applications are to be applied there after.

Treatment for karma of previous birth incarnation. Agasthiyar quoted as follows;

### **கன்மம் தீர**

“தயங்காமல் தீருவதற்கு வகையைக் கேளு  
தன்மையுள்ள வருஷமது தகையுங்காலம்

நயங்காம லாடிய மாவாசையன்று  
நன்மையுள்ள தெளிநீரில் ஷ்நானம் பண்ணி  
முயங்காமற் சிவதலத்தைக் கோரி வந்து  
முத்தியுடனாயிரம் பேர் வேதியர்க்கு  
சுயம்பாகத்தான் கொடுத்து நவக்கிரகந் தன்னைத்  
தோத்தரித்து அட்சதையும் வாங்கிக் கொள்ளே.”

“வாங்கியே பொதிகைக்குத் தென்கிழக்கு முலை  
வரியையுள்ள வானமா மலைக்குச் சென்றால்  
தூங்கியே யாலிலைநேர் பள்ளி கொண்ட  
சுவாமியிட சந்நிதியில் தியானஞ் செய்து  
தேங்கி யேயுதக மென்ற கிணரங் குண்டு  
திரமாக ஒரு செம்பு நெய்யை வாரே”.

“வார்த்துமே மறுசெம்பு தயிலமொண்டு  
மைந்தனே வேடுகட்டிக் கொண்டு போய் நீ  
ஆற்றுமே யந்திசந்தி காசெடையாய்க் கொள்ள  
அப்பனே திங்களது மூன்றுக்குள்ளே  
தோற்றுமே தேகமது பொன்போலே தான்  
துயரமான கன்மனோய் தூரவோடுங்  
காத்துமே யித்த யிலங் கொள்ளவிட்டால்  
கன்மனோய் தீராது யின்னங்களே.”

- செய்யுள் - 217

- Agasthiyar Pari Pooranam 400

Since siddha system of medicine is based on the mukkuṭra theory, the treatment is mainly aimed to bring down the three thodams to its equilibrium state and thereby restoring the physiological condition of various thathus.

## **MODERN ASPECTS**

### **The Skin Anatomy**

The integument, or skin (cutis) is an anatomically and physiologically specialized boundary lamina essential to life. It is major organ of the body, forming about 8% of its total mass and having an area of between 1.2 to 2.2 m<sup>2</sup>. In total thickness ranges from about 1.5 to 4mm. Skin covers the entire external surface of the body including the external auditory meatus and lateral aspect of tympanic membrane. It is continuous with the mucosa of the alimentary, respiratory and urogenital tract at their respective orifices, where the specialised skin of mucocutaneous junction occurs; it also fuses with the conjunctiva at the lacrimal puncta.

Structurally skin is a complex and highly specialized as might be expected of the major interspace between the body and its environment. Microscopically it is formed as an intimate association between two distinct tissues.

Keratinized stratified squamous epithelium superficially the 'epidermis' and a deeper layer of moderately dense connective tissue, the "dermis"

Because of this combination; it is within limits a most effective barrier against microbial invasion and dehydration and against mechanical, chemical osmotic, thermal, photic damage.

## **Epidermis**

The epidermis is formed of non – vascular stratified epithelium. Its usual thickness is between 0.07mm and 0.12 mm. The epidermis is mainly divided into two main systems, they are keratinising (or) malpighian system (keratinocytes) which forms the bulk and the pigmentary system (melanocytes) which produces the pigments.

There are seven layers in the epidermis.

### **Stratum Germinativum (or) Stratum Basale**

This is the deepest portion of the epidermis and is composed of a columnar cells placed perpendicular to the skin surface. The whole of the epidermis germinates from this stratum, hence the name ‘stratum germinativum’

### **Stratum malpighii (or) The Prickle Cell Layer**

It is superficial to the basal cell layer, and is composed of several layers of polyhedral cells connected to each other by intercellular bridges.

### **Stratum Granulosum**

It is superficial to the stratum malpighii. It is composed of flat, fusiform cells which are one to three layers thick. These cells contain irregular, granules of keratohyalin and lysosomal enzymes and cystine rich proteins.

### **Stratum Lucidum**

It is superficial to the stratum granulosum, is the pale, wavy – looking layer. This layer contains refractile droplets of eleidin.

### **Stratum Corneum**

This is the most superficial layer, the outer surface of which is exposed to the atmosphere. It consists of many layers of non – nucleated, flattened and cornified cells.

### **Basal lamina (Basement membrane)**

Dermal Side of the basal lamina contains of few scattered collagen fibres.

## **Dermis**

The dermis which is bounded distally by its junction with the epidermis and proximally by the subcutaneous fat. The basis of the dermis is a supporting matrix (or) ground substance in which polysaccharides, the matrix contains two kinds of proteins. There are

- (a) Collagen - Which has great tensile strength
- (b) Elastin - Which has considerable elasticity.

Hair follicles, various types of sebaceous and sweat glands, plain muscle fibres sensory and organs like pacinian and adipose tissue are seen in the microscopic section of dermis.

The dermis contains few cells, which are fibro blasts, mast cells, histocytes (or) macrophages, lymphocytes (or) other leucocytes and melanocytes. In the deeper layer of dermis, then in arterio – venous anastomosis surrounded by sphincter, the group of smooth muscles under autonomic nervous control.

### **Sebaceous glands**

They are situated in the upper half of the corium. The sebaceous glands are derived from the epithelial cells of the hair follicles and are present everywhere in the skin except on the palms and soles.

They are multilobulated and covered by a connective tissue capsule within, which is a layer of small epithelial cells. As these cells mature towards the centre of the lobules they enlarge, their cytoplasm becoming arranged in a delicate network surrounding globular of fat (sebum). Towards the duct the whole cell disintegrates, lobulating its fat, the glands therefore being classified as a Holocrine glands.

### **Sweat glands**

These are found in all areas of the skin. The sweat glands originate as downgrowths from the dermis. These consist of a single unbranched tube which terminates in the form of a coil in the mid – corium. The coil is the secretory segment and is lined by a single layer of epithelial cells. The duct runs straight upwards from it to the epidermis, which it transverse in a corkscrew manner to open on the surface at the pore. The latter is covered by a loose mesh work of horn cells.

## **Apocrine glands**

They occur in the axillae, areola and nipples of breasts, umbilicus, around the anus and the genitalia. The myo – epithelial cells are highly developed and more abundant in these glands. They are specialized sweat glands, and their secretion is odoriferous with a secondary sexual significance.

## **Hair**

Hair is found on almost every part of the body surface except on the palms and the soles, the dorsal surface of the terminal phalanges, the inner surface of the labia, inner surface of the prepuce and the glans penis. Hair growth and development is under endocrine control.

Hair is made of hard keratin and is analogous to nail. It is formed by the hair matrix, a layer of specified epidermal cells, capping the papilla, the two structures making up the hair bulb. Melanocytes are present in the matrix and form the pigment of hair. The portion of the hair below the surface of the scalp is known as the hair root. Above the surface of the scalp is known as the hair shaft. Above the surface of the scalp, the hair is composed of the medulla, cortex, cuticle. The medulla consists of seven rows of soft keratin, but it is discontinuous or even absent in most human hairs. The cortex is the main structural component and is made up of tightly packed fusiform keratinized cells.

## **Nails**

These are semi – transparent, plates – like horny structures, covering the dorsal surfaces of the distal phalanges of the fingers and toes. The nail is composed of many layers of flattened keratinized cells fused into a homogenous mass.

They arise from epidermis lining an invagination of skin at the base of the nail, this specialised epidermis known as the nail matrix. The invagination of skin at the base of the nail is called the nail fold. The anterior border encroaches upon the nail plate as a flattened keratinous rim, the cuticle and forms a protective barrier against irritants and infection.

## **Blood Vessels**

The blood supply of the skin originates from a large number of arterioles forming anastomosis in the deepest part of the cortex. From here single vessels run upwards and form a second network in the upper cortex. Finally terminal arterioles ascend into the papillae ending in capillary loops; which drain into connecting venules. The blood is returned to the large veins in the subcutaneous tissue.

## **Lymphatics**

The skin contains a rich network of lymphatics, which drain into a few larger vessels in the hypodermis.

## **Nerve Supply**

The nerve supply of the skin consists of a motor sympathetic portion derived from the sympathetic ganglia and sensory spinal portion arising from the dorsal root ganglia. The sympathetic fibres innervate the blood vessels, erector pili muscles and apocrine duct, where the fibres are adrenergic and cause contraction.

## **PHYSIOLOGY**

### **1. Protective Function**

The epidermis and subcutaneous fat play roles in the protective functions, the mechanical properties of the skin depends mainly on the dermis. It protects the penetration of harmful substances and bacterial invasions. Another is to protect against sunlight by synthesis of melanin pigment.

### **2. Immunological Function**

The skin is the front line of the defences of the body. In essence the defence involves the protection of antibody – complexes, multi hair proteins which bind with the offensive antigens. Langerhan cells probably play a crucial role in the contact sensitization, surveillance against viral infections and neoplasms.

### **3. Sensory Functions**

The skin is richly supplied with nerves and various types of specialized sensory end – organs, which provide information regarding environmental changes, so that the body can then adjust its activities accordingly. In some animals, the hair at certain situations have



specialized sensory receptors located at the bases of the hair follicles which serve to enhance sensory appreciation.

#### **4. Secretion and Excretion**

The skin possesses various types of glands, which pour secretions on the surface. The more important glands are sweat and sebaceous glands. The eccrine glands which are scattered all over the body surface secrete a thin, transparent watery fluid, known as true sweat; while the apocrine glands secrete a thicker, rather milky and odoriferous solution.

Sweat in its composition consists of 1.2% solids and 98.8% water. The important substances excreted in it are sodium chloride, sodium phosphate, sodium bicarbonate, urea and a small amount of urea. The skin can also excrete certain drugs administered to the individual, for example mercury, arsenic, iodine etc.

The sebaceous glands of the skin secrete sebum, which is composed of fatty acids, cholesterol, alcohols etc. fatty acids have a mild fungistatic activity. The sebum acts as a lubricant for the drying for the drying effects of the atmosphere.

#### **5. Synthesis of Vitamin D**

Vitamin D is synthesised in the skin as a result of exposure to ultra violet 'B' (UVB) radiation and, since it is carried in the blood attached to a binding protein to exercise a specific effect at a different sites. Vitamin D<sub>5</sub> is essential for skeletal development, and it contains antirachitic properties. Vitamin D<sub>3</sub> is formed principally in the stratum spinosum and the stratum basale, from the precursor 7-dehydrocholesterol by way of a provitamin D<sub>3</sub> (2,5).

#### **6. Body Heat Regulation**

The skin plays the most important role in the regulation of heat loss. It loses heat to the external environment in three ways: by conduction, by convection, by radiation and by evaporation. Heat loss by the first two mechanisms take place when the environmental temperature is lower than that of the skin. Heat loss by evaporation mainly means the amount of heat spent by the body to evaporate the sweat from the surface of the skin. About 90% of the total heat loss of the body is regulated by the skin. The heat loss through the skin is regulated by various physiological mechanisms which include

1. The reaction of the cutaneous vessels.

2. the reaction of the smooth muscle fibres of the skin and
3. perspiration.

### **7. Endocrine Function**

Hair follicles and sebaceous glands are the targets of androgenic steroids secreted by the gonads and the adrenal cortex and melanocytes and directly influenced by polypeptide of the pituitary.

### **8. Storage Function of Skin**

Blood is stored in the rich sub papillary plexus of the dermis, about one litre. The skin is also a good store house of ergosterol which is irradiated, by the ultra violet light of the sun and converted into vitamin D.

The junction between dermis and hypodermis has a considerable capacity for storing fat and permanent store of subcutaneous adipose tissue. Certain substances like glucose and chloride also acts as a reservoir for topically applied corticosteroids (or) other hormones which absorb slowly for many days from the skin surface.

### **9. Absorption**

The skin can absorb substances dissolved in fatty solvents like vitamins and hormones. Inflammation greatly increases the skin permeability substances that are completely insoluble in water and lipids do not penetrate.

### **10. Gaseous exchange through Skin**

A small amount of gaseous exchange occurs through the skin. In man the amount of  $\text{CO}_2$  exchanged through the skin is negligible compared to the amount exhaled from lungs.

## **AUTO IMMUNE DISORDERS**

Auto immunity is a condition, in which structural or functional damage is produced by action of immunologically component cells or antibodies against normal components of body. Auto immunity literally means “Protection against self” and therefore it has been criticised as contradiction in terms.

In auto immune disorders the body forms antibodies against its own tissues or arms T cells to attack them. The abnormal immune response is sometimes due to a derangement of immune system, sometimes to an alteration in tissue attacked.

Antibodies form against these antigens and T cells become sensitized to them but the reaction does neither good nor harm

Occasionally, an autoimmune reaction is beneficial. One theory suggests that tumour cells are destroyed by immune responses against abnormal antigens on their surface before they can grow into a big enough to be recognised. It is usually believed that destruction is essential if the body is to be cleared of virus.

Other autoimmune reactions cause serious injury. The destruction of red cells by antibodies that fix complement in the autoimmune anaemia the hyperthyroidism caused by the attachment of an antibody to the cells of the thyroid gland in Graves disease, the glomerular basement membrane in Goodpasture's syndrome and the many other conditions in which immune system plays an important role.

### **Mechanism of autoimmunizations**

1. Clonal elimination
2. Suppressor cells
3. Blocking antibodies
4. Anti-idiotypic network control

#### **Pathogenesis of Autoimmunity**

The mechanism by which the immune tolerance of body is broken causes autoimmunity. These mechanisms may be immunological genetic and microbial all of which may be interacting.

#### **1. Immunological factors:**

Failure of immunological mechanism of tolerance initiates auto immunity.

#### **These mechanisms are as follows:**

1. Polyclonal activation of B cells.
2. Generation of self-reacting B cell clones.
3. Decreased T suppressor and increased T helper cell activity.
4. fluctuation of anti-idiotypic network control.

5. Sequestered antigen released from tissues.

## **2. Genetic Factors**

1. There is increased expression of class –II HLA Antigens on tissues involved in auto immunity.
2. There is increased familial incidence of some of auto immune disorders.

## **3. Microbial factors**

4. Particularly virus (eg. EBV infection) and less often bacterial (eg: Streptococci, Klebsiella) and mycoplasma.

Types and examples of Autoimmune diseases

### **1. Organ specific diseases**

In these the auto antibodies formed react specifically against an organ or target tissue component and cause its chronic inflammatory destruction. The tissues affected are endocrine glands (thyroid, pancreatic islets of langerhans, adrenal cortex), alimentary tract, blood cells and various other tissues and organs.

### **2. Non organ specific diseases**

These are disease in which a number of auto antibodies are formed which react with antigens in many tissues and thus cause systemic lesions.

Non organ specific (systemic)

- |                                   |                                    |
|-----------------------------------|------------------------------------|
| 1. Systemic lupus erythematosus   | 5. Poly arteritis nodosa (PAN)     |
| 2. Rheumatoid arthritis           | 6. Sjogren's syndrome              |
| 3. Scleroderma                    | 7. Reiter's syndrome               |
| 4. Polymyositis – Dermatomyositis | 8. Mixed connective tissue disease |

organ specific (Localised)

1. Hashimoto's (auto immune) thyroiditis
2. Graves disease
3. Insulin dependent diabetes mellitus
4. Idiopathic Addison's disease

## **2. Alimentary Tract**

1. Auto immune atrophic gastritis in pernicious anaemia
2. Ulcerative colitis

3. Crohns disease

### **3.Blood cells**

1. Auto immune haemolytic anaemia.
  2. Auto immune thrombocytopenia
  3. Others
- |                                  |                                  |
|----------------------------------|----------------------------------|
| 1. Myasthenia gravis             | 5. Primary biliary cirrhosis     |
| 2. Auto immune orchitis          | 6. Chronic active hepatitis      |
| 3. Auto immune encephalomyelitis | 7. Membranous glomerulonephritis |
| 4. Good pasture's syndrome       | 8. Auto immune skin disease      |

Diseases of anti immune origin usually exhibit the following features:

1. An elevated level of immunoglobulins
2. Demonstrable auto antibodies
3. Deposition of immunoglobulins or their derivatives at site of election such as renal glomeruli.
4. Accumulative of lymphocytes and plasmsa cells at the sites of lesions.
5. Temporary or lasting benefit from corticosteroid or other immuno suppressive therapy.
6. the genetic predisposition auto immunity.

## **PSORIASIS**

### **Definiton**

It is a common, chronic and non-infectious skin disease characterized by well-defined slightly raised, dry erythematous macules with silvery scales and typical extensor distribution.

## **Synonyms**

The designation “Psoriasis” has been preferred for many years and there are no synonyms of importance.

## **Basic facts about the Genetics of Psoriasis**

Among, the benefits expected to derive from the Human Genome Project is the identification of specific genes associated with specific diseases. While there is always hope that identification will be simple as “one gene of disease” this is unlikely to be the case for most major diseases that have genetic component. For most diseases including psoriasis there are probably multiple genes involved in producing the sequence of events that results in the expression of disease complicating the picture still further is the probability that genes found to be associated with psoriasis may or may not cause psoriasis in an individual person, depending on the activity of the genes in relation, to one another. The activity of genes in relation to one another is often determined by the pattern in which they were inherited from parents.

## **Psoriasis a disease with Genetic Predisposition**

Numerous studies over many years support the finding that genetic predisposition an inherited tendency to develop the disease has a major role in the pathogenesis of psoriasis. Genetic predisposition does not mean a 100 percent guarantee that the disease, will appear; other initiating factors such as injury or infection may act together with genetic predisposition to the disease process in motion supporting evidence for genetic predisposition includes

- There is a higher – than – average incidence of psoriasis in relatives of people with psoriasis, indicating “familial tendency” to develop the disease; however in some people with psoriasis no family history is evident.
- There is an increased incidence of psoriasis in children when one or both parents has psoriasis.

- In studies of identical and non identical twins. Psoriasis is much more likely to appear in both identical twins than in both non identical twins a finding that also confirms that more than one gene must be inherited to establish predisposition for psoriasis.
- There is higher than expected frequency of certain white cell antigens (class I human Leucocyte Antigens or HLA's ) on cells of people with psoriasis and their close relatives; this finding also supports psoriasis inheritability and also suggests that the gene(s) involved in psoriasis may be on the some chromosome that holds the genes for HLA. There are many types of HLA in the HLA complex and studies have shown that HLA type may be associated in some degree with timing of disease onset, type of psoriasis and diseases severity.

### **Sites of Predilection**

The lesion in ordinary case have a predilection for the scalp, nails, extensor surfaces of the limbs ( especially the shins ) the elbows, the knees and the sacral region.

### **Age of onset**

The onset of Psoriasis constitutes a Lifelong threat. As demonstrated by several studies most patients develop the initial lesions of psoriasis in the third decade of life. First signs appear in males at mean age of 29, and in females at age 27, a recent study of the onset of psoriasis in 2400 patients showed a peak incidence at 22.5 years of age; a second peak of onset around the age of 55 was found in 11.8 percent of the patients.

## **Epidemiology**

### **Incidence**

Psoriasis is universal in occurrence. In the United States Psoriasis affects about 1 percent of the population: however the world wide incidence varies considerably. Reasons for such variations range from racial to geographic and environmental. Psoriasis is equally common in males and females.

## **Historical Aspects of Psoriasis**

The earliest descriptions of what appears to represent psoriasis are given at the beginning of medicine in the corpus Hippocraticum. This work was edited in Alexandria 100 years after the death of Hippocrates (460- 377 B.C) who presumably was the author. Hippocrates used the terms **Psora** and **lepra** for conditions that can be recognized as Psoriasis.

## **Pathogenesis:**

Accelerated epidermopoiesis has been considered to be the fundamental pathologic event in psoriasis. The transit rate of psoriatic keratinocyte is increased and the deoxyribo nucleic acid synthesis time is decreased. It has been suggested that it is the heightened proportion of epidermal cells participating in the proliferative process rather than the actual rate of epidermopoiesis, that is the basic fault in psoriatic lesions. The result in either case is greatly increased production of keratin.

The earliest histologic change was inflammatory perivascular upper dermal infiltrate, with only epidermal acanthosis and parakeratosis after the transformation of the lesion into a scaly papule.

Polyamines are significantly increased in Psoriatic lesions. There is an increased production of leucotrienes and 12-hydroxy eicosatetraenoic acid, both of which are chemotactic for polymorphonuclear leukocytes.

## **The Arachidonic acids cascade:**

The complexity of the pattern of inducers of inflammation is now much better shown than in the production of leucotrienes, prostaglandins and eicosatetraenoic acid from arachidonic acid. Arachidonic acid is obtained from dietary sources; membranes of red meat, green leafy vegetables and corn and sunflower oils. Its products are central to the aggravation of Psoriasis by trauma and to the pathogenesis of psoriatic lesions.



Psoriasis is associated with different HLA antigens. They are B<sub>13</sub> or B<sub>17</sub> has a five fold risk of developing psoriasis. In pustular Psoriasis HLA-B<sub>27</sub> may be seen whereas B<sub>13</sub> or B<sub>17</sub> are increased in guttate and erythrodermic psoriasis. In palmoplantar psoriasis, there is an increased proportion of persons having HLA-B<sub>8</sub>, Bw, 35-Cw7 and DR3.

Farber affirms an abnormal nucleoprotein metabolism in the incomplete Keratinization process in psoriasis. In the stratum corneum the free amino acids are low, with an accumulation of MPS and of free and esterified choline. DNA and RNA accumulate and pentose, purins, uracil and organic phosphates are also increased.

There is prominent neutrophil response in psoriatic lesion. How this relates to findings of increased plasminogen activatory activity in psoriatic skin or to the enhanced physiologic functions that have been attributed to psoriatic polymorpho nuclear leucocytes or to the immunologic findings of IgG and complement in the stratum corneum is not yet known. Both psoriasis and Reiter's disease occur with increased frequency in patients with AIDs. Also interleukin – 2 therapy for malignancy may induce psoriasis by what mechanism is not clear (Reference – Text book of Dermatology – fitz patrik)

Weddell showed that there is a profuse hyperplasia of nerve endings beneath and into the lesion of psoriasis.

## **Pathology and pathogenesis**

The histopathological appearance of psoriasis is distinctive but not specific. The main features may be subdivided into (1) the epidermal thickening, (2) the inflammatory component and (3) the vascular component, but of course all are closely interlinked.

### **The epidermal thickening:**

The epidermis shows marked exaggeration of the rete pattern and elongation of the epidermal down growths with bulbous club like enlargement of their ends. The average thickness is increased from about three to four cells in the normal skin to approximately 12-15 cells in the psoriatic lesion. Many mitotic figures can be seen and the rate of epidermal cell production seems to be greatly enhanced when epidermal cells

are labelled in DNA synthesis by exposing them to a radiolabelled precursor of DNA in vitro or in vivo and an auto radiographic technique to demonstrate the labelled cells, there is an obvious increase of cells in division in psoriasis compared to normal. The turnover time of psoriatic epidermis and stratum corneum is consequently very much shortened. Normally it takes some 28 days for newly born cells to ascend from the basal layer and travel through the epidermis and the stratum corneum and reach the surface and desquamate off. In psoriasis it takes some four days!

Surmounting the thickened epidermis is a stratum corneum in which epidermal nuclei have not disappeared during differentiation ('parakeratosis')

### **The inflammatory Component**

Interpersed between the 'parakeratotic' horn cells are collections of desquamated polymorphonuclear leukocytes known as Munro microabscesses. The epidermis is oedematous and itself infiltrated by inflammatory cells. The dermis immediately below the epidermis also contains many inflammatory cells, mostly lymphocytes. In pustular psoriasis the epidermal component is much less in evidence and there are collections of inflammatory cells within the epidermis. It has been suggested that chemotactic influences attract the inflammatory cells into the epidermis and stratum corneum and both complement components and eicosanoids – particularly leukotriene B<sub>4</sub> – have been identified in psoriatic stratum corneum.

### **The Vascular Component**

The papillary capillaries are greatly dilated and tortuous to a degree not seen in other inflammatory skin disorders. Ultrastructurally it can be seen that there are larger gaps than usual between the endothelial cells.

These abnormal capillaries are the last of the features to go during resolution and it has been claimed that abnormal capillaries can be detected in the apparently normal uninvolved skin of psoriatic subjects.

## **Cell systems involved in pathogenesis of Psoriasis**

### **Keratinocytes;**

A characteristic feature of involved skin of psoriatic subjects is hyper proliferation. There is two fold increase in proliferative cell population and 100 percent of the germinative cells of the epidermis appear to enter the growth fraction compared with 60 to 70 percent for normal subjects.

### **T cells**

Numerous T cells are present in psoriatic lesions predominantly surrounding the vessels of the upper dermal plexus.

### **Granulocytes**

Formation of spongiform microabscesses (Munro micro abscesses) filled with granulocyte is a hallmark of psoriasis. The presence of these cells in psoriatic lesions is variable and becomes more pronounced with disease activity (eg) in acute guttate or pustular psoriasis.

### **Resident cells**

#### **Endothelial cells**

Changes in the dermal capillary endothelium have repeatedly been implicated at the site of primary defect. Hyper proliferation of endothelial cells is most pronounced in the tortuous dilated capillaries at the advancing edges of a lesion.

#### **Mast cells**

Mast cell densities are increased in lesional psoriatic skin compared with normal or uninvolved psoriatic skin.

#### **Fibroblasts**

Fibroblasts are potent producers of cytokines and lipid mediators that could affect the epidermis as well as the inflammatory reaction.

## **Terms Describing Morphologic Features of Psoriasis**

### **Psoriasis Ostracea**

Old patches may be thickened tough and covered with lamellar scales like outside of an oyster shell.

### **Psoriasis gutata**

The lesions are the size of water drops.

### **Psoriasis follicularis**

Tiny scaly lesions are located at the orifices of the pilosebaceous follicles.

### **Psoriasis figurata, Psoriasis annulata & Psoriasis gyrata**

The lesions have curved linear patterns produced by central involution.

### **Psoriasis Discoidea**

In this type central involution does not occur and solid patches persists..

### **Psoriasis rupoides**

In this type crustaceous lesions occur resembling syphilitic rupia.

### **Psoriasis Flexura**

Better known as inverse psoriasis and is found in intertriginous area.

### **Volar Psoriasis**

Seen in palms and soles

## **Trigger factors for Psoriasis**

External factors may provoke manifestation of psoriatic skin lesions, they may be called trigger factors.

Natural incidence studies suggest that environment plays a predisposing role. Hot weather and sunlight are reported to be beneficial, whereas cold weather appears to have an opposite effect.

### **Physical trauma – Koebner's Phenomena**

In 1872, a Dr. Koebner described a patient with Psoriasis who developed new lesions at spots where he was bitten by his horse. The relationship between physical trauma and psoriatic lesions was subsequently documented in many more patients and was given the name "Koebner's phenomenon". An analysis of patient record has indicated that up to 50 percent of persons with psoriasis have a psoriatic lesion develop at the site

of an injury or skin condition Koebner's phenomenon is probably increased during an active phase of the disease.

### **Infections**

The frequency with which infections trigger psoriasis varies from a low of 15 percent to a high of 76 percent. Children report the onset of psoriasis after clinically recognized infections, particularly upper respiratory infections.

Acute guttate psoriasis frequently follows an acute streptococcal infection by 1-2 weeks. Streptococcal infections may play a role in exacerbating other forms of psoriasis.

Infection with the Human Immuno Deficiency Virus type (HIV I) may represent another important trigger factor, although the incidence varies considerably.

Staphylococcal skin infection (boils) have been a trigger).

### **Stress**

It is difficult to separate job and family related stress from the psychological stress of living with psoriasis. One cause of stress probably reinforces the other. Nevertheless the perception of patients that psychological stress can worsen psoriasis has been supported in clinical studies. Patients may be tempted to reduce stress by abusing drugs or alcohol – activities that can actually increase stress. A stress depression, stress aggression or stress obsession pattern may be accompanied by increased pruritus (itching). A psoriasis patient who feels overwhelmingly stressed should ask the dermatologist to arrange for psychological counselling, which may include members of the family.

### **Anatomic Sites**

Certain anatomic sites are prone to develop disease and such can be considered within the realm of trigger factors. In chronic stationary psoriasis the scalp is most frequently involved, followed by the knees and elbows. In guttate psoriasis of acute onset the trunk and proximal extremities are predominantly affected.

### **Drugs**

Psoriasis has been reported to follow rashes caused by ingestion of drugs. It is also now recognized that systemic corticosteroids possibly some topical corticosteroids will upon withdrawal after prolonged use, frequently result in a severe flare of psoriasis.

The reported exacerbations of psoriasis associated with antimalarials may have been over emphasized in the past. Lithium is a known inducer of psoriasis and can cause

exacerbation of existing psoriasis. Beta adrenergic blockers have caused psoriasis like eruption and a flare of psoriasis as well.

## **Types of Psoriasis**

### **Common Plaque or Nummular Psoriasis**

The commonest form of the disease is the plaque or nummular variety in which round or oval lesions of one to may centimeters are found on the elbows, knees, scalp and trunk in insidious onset. This type of psoriasis may remain stationary for long periods of time; Exacerbations may bring small guttate lesions along with the large plaques serpiginous, annular, gyrate and zonal arrangement of lesions may be seen along with koebner – induced linear papules produced by scratching. Thick, inveterae, plaques may occur in patients with very chronic involvement of the elbows, knees or hips.

### **Guttate psoriasis**

The abrupt appearance of a shower of small, drop like psoriasis lesion with a less prominent scale than usual over much of the skin surface, but especially on the trunk and proximal extremities, should suggest this special syndrome. It is seen primarily in children and young adults and commonly occurs a few weeks after streptococcal infections. Elevated antistreptolysin ‘O’ titer are usually found. The lesions often gradually disappear after resolution of the infection.

### **Palmar Psoriasis**

A distinctive, patchy, hyperkeratotic type of psoriasis principally affecting contact points of the volar surface of the fingers and palms this form of the disease may exist alone or in combination with mild psoriasis, usually of the elbows, knees and scalp. The lesions are related to local physical or chemical injury and are thus volar koebener phenomena. It may involve on palm more than the other as in tennis players, golfers or industrial workers, strangely, the same changes are less common on the soles. Palmar psoriasis is often confused with chronic contact dermatitis. It runs a protected course of a few to several years, but may persist indefinitely. Palmar psoriasis is to be distinguished from pustular psoriasis of the palms and soles.

### **Psoriasiform neurodermatitis**

Chronic psoriatic plaques may intensely, provoking considerable scratching and a lichenoid appearance.

A rather common syndrome, however, is the lichenoid neurodermatitis on the occipital scalp and nape of the neck, which develops a psoriasiform appearance. The majority of patients with this condition, usually women, have no psoriasis elsewhere, and would best be regarded as having a primary neurodermatitis rather than psoriasis.

### **Pustular Psoriasis**

There are two clinically dissimilar conditions to which the name pustular psoriasis has been applied, namely:

1. Localised Pustular psoriasis
2. Generalised pustular psoriasis

#### **Localised pustular psoriasis**

Also known as pustular psoriasis of the palms and soles, this sterile pustular eruption affects the digits and volar skin of the hands or feet, or both usually in the presence of papulo squamous psoriasis elsewhere.

In its commonest form, superficial lakes of pus appear in crops on a background of erythema, most commonly on thenar or hypothenar eminences or centre or inner aspect of the sole.

The individual pustules dry up, turn brown and exfoliate off, but the whole condition is very chronic and lasts years or decades.

#### **Generalised pustular psoriasis**

Pustulation may become widespread and associated with sheets of vivid erythema. This is generalised pustular psoriasis. This is the severest form of psoriasis and is fatal in about a quarter of cases.

### **Flexural Psoriasis**

This type of psoriasis occurs mostly in the body folds – such as groins, axillae and infra – mammary regions. Seen in middle – aged obese individuals women rather than men. The lesions are well demarcated, with reduced scaling, frequently itch.

## **Psoriasis and Bullous Pemphigoid**

The co – existence of psoriasis and bullous pemphigoid has been described in at least 28 cases in the literature. In many of these patients the bullous pemphigoid was believed to have been precipitated by treatment of the psoriasis with topical anthralin. Crude coal tar, salicylic acid, ultraviolet light (UVL), sunlight of Psoralene Ultra Violet Administration (PUVA)

## **Psoriasis Arthropathica**

In this form, psoriasis is associated with a type of erosive arthritis, very similar to Rheumatoid arthritis.

### **Exemption:**

a). Distal Interphalangeal joints were affected in more than 50% of cases.

a. Rose – waaler test negative in over 90% of cases.

The nails were also involved in over 90% as compared to the usual average of 50%

Joints of fingers, feet, ankles, knees and sacro –iliac are selectively affected; these joints are swollen and painful. The psoriatic eruption & involvement of the joints may increase or decrease simultaneously.

## **Psoriatic Nail Diseases:**

Nail involvement is common in psoriasis. Nails are involved in about 50% cases. Finger nails show changes more often than the toe nails.

Nails show 3 types of lesions;

- a. They may show multiple small pits like those on a thimble.
- b. They may become detached from its bed over the distal half of the affected portion being opaque friable and discoloured.
- c. They may become thickened rough and pigmented.

It is important to realise that psoriasis does not produce the destructive cicatricial change of lichen planus, which may result in a band like pterygium or loss of the nail.

Acute fulminant psoriasis or pustular psoriasis affecting the paronychia and proximal nail fold skin may also result in nail dystrophy.



Classic psoriatic arthritis affecting the distal interphalangeal joints, is usually associated with nail dystrophy. Nail involvement in the absence of psoriatic skin lesions is rare and may be difficult to prove.

The thickened or onycholytic psoriatic nail or subungual tissue frequently contains *Candida albicans* and *Pseudomonas* organisms. The latter may at times produce a greenish discolouration of the nails. The psoriatic nail is, however, peculiarly resistant to invasion by superficial fungi.

### **Napkin Psoriasis**

Napkin Psoriasis or psoriasis in the diaper area may also be seen in infants between two to eight months of age. It probably starts off with irritation of the skin in the area and treatment should once again be directed to better hygiene and care of the skin under the napkin.

### **Systemic Association of psoriasis**

#### **Inflammatory Bowel Disease**

The strong linkage of HLA – B27 to ankylosing spondylitis and ulcerative colitis and the increased frequency of this haplotype in patients with psoriasis and arthritis (about six times normal ) suggest that ulcerative colitis should be seen more frequently in patients with psoriasis. The frequency of psoriasis among patients with ulcerative colitis and Crohn's disease is respectively, 3.8 and 7.6 times normal.

#### **Occlusive Vascular Disease**

Abnormalities in the microvascular components of the involved and uninvolved skin of patients with psoriasis have caused investigators to propose an association between psoriasis and large vessel disease. Current evidence suggests that patients with psoriasis especially males, have an increased incidence of occlusive vascular disease (thrombophlebitis, Myocardial infarction, pulmonary embolism and cerebral vascular accidents)

**Systemic Effects of psoriasis** Generalized pustular Psoriasis as described by Von Zumbusch, is the form associated with systemic findings. This form of psoriasis appears as waves of sterile pustules on an erythematous skin, characteristically, short episodes of fever 39°C to 40°C are followed by another wave of new pustules. In addition to fever

there are systemic signs of disease such as weight loss, muscle weakness, leucocytosis, hypocalcemia and an increased sedimentation rate. In patients with pustular psoriasis, arthropathy is common as is the HLA – B27 haplotype.

#### Association with Pregnancy

Studies of approximately 500 patients suggest that patients with psoriasis have a normal risk of systemic cancer and possibly increased risk of cutaneous cancer.

### **Diagnosis of Psoriasis**

The diagnosis of psoriasis is based upon:

1. The family history of psoriasis.
2. The typical distribution of lesions on the scalp, elbows, knees, the front of the legs, back and nails.
3. Well – defined, non – indurated dry, erythematous areas with silvery, layer – upon – layer scaling.
4. The candle – grease sign (When a psoriatic lesion is scratched with the point of a dissecting forceps, a candle – grease – like scale can be repeatedly produced even from the non – scaling lesions. This is candle – grease sign.
5. Auspitz sign (Complete removal of a scale found pin – point bleeding)
6. Koebner's phenomenon  
(Psoriatic lesions may develop along the scratch lines in the active phase)
7. Little or no itching
8. History of previous attacks, and seasonal variations of the disease
9. Typical histopathology

The above – mentioned histopathological findings are not specific for psoriasis alone. There are no laboratory tests which will positively identify psoriasis. The blood count, urine analysis, ESR, and other haematologic, chemical and serologic studies are within normal limits in most cases of psoriasis.

### **Complication of Psoriasis**

1. Complications are infrequent. Psoriasis arthritis can cause disability.
2. Exfoliative dermatitis
3. Eczematous lesion caused by scratching and infection.
4. Lichenification brought by scratching

## **Prognosis**

A permanent cure is not yet more, but individual attacks can be controlled satisfactorily. General health and longevity are unaffected. The clinical course of the lesion is chronic with various periods of remissions (weeks to years). The whole position would be explained to the patient. He should be encouraged for persisting towards the treatment until the lesions have disappeared. Psoriasis does not leave scars. The nail gradually assumes the normal appearance. The palmar and nail psoriasis are more resistant to the treatment.

## **Common Laboratory Abnormalities in Psoriasis**

1. Elevated uric acid
2. Mild anemia
3. Negative nitrogen balance
4. Increased sedimentation rate
5. Increased  $\alpha_2$  Macro globulin
6. Immuno globulin aberrations, increased IgA levels and increased quantities of immune complexes.

## **Serum Uric acid**

Serum uric acid is elevated in 30-50 percent of patients with Psoriasis. This increase is thought to be caused by the increased epidermal proliferation seen in psoriasis and it is associated with break down of DNA. Elevated Uric acid levels increase the risk of an attack of gouty arthritis and there are reported cases of typical gouty arthritis with Psoriasis.

## **Haematologic findings**

### **Folate metabolism;**

It is not uncommon for patients with psoriasis to present with mild anemia. Although the anemia is usually categorized as anemia of chronic disease, there is evidence of folate and iron abnormalities.

### **Iron Metabolism:**

Iron content in normal stratum corneum is 26  $\mu\text{g/g}$  and the normal loss of stratum corneum per day approaches 1g. In psoriasis the stratum corneum of involved sites in

patient's with psoriasis is two times normal. These calculations suggest that up to 2.5 mg of iron can be lost per day via desquamation.

### **Protein Loss;**

Negative nitrogen balance, defined as protein loss exceeding nutritional requirements, may be reflected in serum albumin levels. Although the pathologic significance is unknown, hypoalbuminemia has been noted in patients with severe psoriasis.

### **Serum Proteins:**

Patients with Psoriasis have increased levels of C-reactive protein and  $\alpha_2$  macro globulin and generally their sedimentation rates are elevated recently it has been observed that serum IgA levels and IgA immune complexes are elevated in patients with psoriasis.

## **Differential Diagnosis of Psoriasis**

In the majority of cases, the diagnosis of psoriasis is usually easy if the above mentioned features are borne in mind. A typical case may create diagnostic problems. The following conditions must be particularly considered in differential diagnosis.

### **1. Seborrhoeic dermatitis**

The scalp patches are diffuse, ill – defined and moist; the hair is matted and tangled in the curls; the crusts are greasy. Body lesions affect the flexures, the sternal and inter – scapular regions. Sebo – psoriasis is a condition in which features both of psoriasis and seborrhoeic dermatitis are seen as indistinguishable.

### **2. Syphilitic Psoriasis**

The history reveals an illicit exposure and the development of chancre; the rash is less scaly, and shows some degree of induration, mucous patches and lymphadenopathy. The V.D.R.L. is positive.

### **3. Pityriasis rosea**

A short history, centripetal distribution, a herald patch and typical oval lesions with cigarette-paer like, centripetal scaling

The flexural lesions must be distinguished from those in tinea cruris, intertrigo, seborrhoeic dermatitis; the nail lesions, from the lesions in tinea unguium, eczema, paronychia and syphilis; the palmar lesions, from the other causes of

hyperkeratosis; the guttate variety, from lichen planus , and the erythroderma type from the causes of erythroderma.

#### **4. Dandruff**

The edges of psoriatic patches are sharp, dandruff patches are indefinite. The scales of psoriasis are dryer and more silvery than those of dandruff. The scales are moist and yellow.

#### **5. Ring worm:(Tinea circinata)**

May resemble circinate psoriasis. But ring worm as a much greater tendency to show pustules or vesicles. The fungus can generally be found microscopically.

#### **6. Lichen Planus**

A careful examination made in a good daylight will reveal the violaceous. The colour lichen planus as opposed to the red brown colour of psoriasis. In lichen planus it is possible to find the characteristic polygonal, flat topped, shiny papules, on the front of the fore arm or wrist. Small white spots may be found inside the cheeks in about 50% of the cases. Lichen planus also itches more than psoriasis.

The flexural lesions must be distinguished from these in tinea cruris, intertrigo, seborrhoeic dermatitis; the nail lesions, from the lesions in tinea, onychomycosis, eczema, paronychia and syphilis; the palmar lesions, from the other causes of hyperkeratosis; the guttate variety, from lichen planus, and the erythroderma type from the other causes of erythroderma.

# PSORIATIC ARTHRITIS

Psoriatic arthritis is an apparently autoimmune inflammatory disease involving the ligaments, tendons, fascia and joints. 5 to 8 percent of persons with all types of psoriasis but occurs at higher frequencies in those with more severe skin involvement, and especially in those with pustular psoriasis.

Psoriatic arthritis is characterized by a relatively greater involvement of joints of the upper limb, especially the hands. Pustular psoriasis with psoriatic arthritis is closely related to Reiter's syndrome.

## Immuno Pathogenesis

1. Initiation of specific process by an antigen recognition even MHC class I molecules present endogenous antigen to CD8 lineage T Cells.
2. Elaboration of cytokines by the largely CD 8 driven immune response.
3. Amplification mechanisms through cytokine and growth factor – induced modulation of phenotype of cells involved in immune response leading.
4. To extensive non antigen – mononuclear cell infiltration of subsynovial lining tissues.
5. Metaplastic alteration of phenotype and growth properties of synovial lining cells and synovial fibroblasts secondary to the cytokines and growth factors.
6. Further amplification by interaction between synovial cells releasing cytokines and infiltrating mononuclear cells with appropriate receptors.

## Pathology

### Synovitis

The central lesion within the joint is synovitis, which resembles the same chronic inflammatory process found in a variety of arthritides from rheumatoid arthritis to Lyme disease. There is metaplastic proliferation of the synovial lining cell layer that converts it from a single into a multi vessel formation stimulating a granulomatous reaction. The early lesions in the synovium are characterized by inflammatory oedema with fibrin deposition and progressive hyperplasia of the lining cell layer. Inflammation and destruction are two additional features characteristic of psoriatic arthritis. Combination

of fibrosis and calcification in joints is not significantly evident in rheumatoid arthritis and is a useful point of distinction.

The synovial fluid in psoriatic arthritis may have modest and variable levels of neutrophils ranging from 1000 to 15000 per  $\text{mm}^3$ . It has a complement level that is elevated above normal when adjusted for protein concentration because of the combination of joint inflammation and elevated serum complement levels that reflect systemic inflammation.

### **Enthesopathy**

Enthesopathy is characterized by an accumulation of lymphocytes and monocytes at the site of tendon or ligament insertion, with consequent erosion and reactive new bone formation that results in spurs and periostitis. The development of the “sausage” digit of dactylitis is based on extensive periarticular inflammation and associated oedema primarily localized to the tendons and ligaments of the digit and principally affects their sheaths and insertion sites.

## **Clinical Manifestations**

### **Onset and Occurrence**

Psoriatic arthritis characteristically develops between ages 35 and 45 but can occur at nearly any age. The onset is usually insidious but in less than one third of persons it is abrupt. Constitutional features including fever and malaise are rare and usually seen only in a fulminant onset with widespread joint disease although the Erythrocyte sedimentation Rate and the serum complement level are commonly elevated to reflect activation of acute phase reactants by cytokines.

### **Skin Diseases**

Susceptibility to the development of arthritis generally increases with the severity of cutaneous involvement. In most persons considered to have Psoriatic arthritis, however about one in seven instances the arthritis develops first. In some persons skin involvement is minimal and it is important to look for subtle lesions such as behind the ears, the buttock, cleft or a few pits in the nails.

## **Joint Disease**

The pattern of peripheral joint involvement and the associated findings of nail disease tenosynovitis, enthesopathy and sometimes axial involvement provide hall mark for the recognition of Psoriatic arthritis.

### **Peripheral Joint Disease**

More than 80 percent diagnosed with psoriatic arthritis manifest a peripheral asymmetric oligoarthritis that involves small joints of hands and feet, the large joints of the legs or a combination of both small and large joints. Inflammation of one or many of the distal interphalangeal joints. Inflammation of one or many of the distal interphalangeal joints is virtually pathognomonic for psoriatic arthritis – Ankylosis of one or more isolated joints is another distinctive feature. The large joints that are affected in psoriatic arthritis include the hips, knees and ankles.

In approximately 15 percent of persons with psoriatic arthritis the pattern of joint involvement in the hands and feet exhibits a much more symmetric character. Psoriatic arthritis can be identified by the presence of more subtle evidence of imperfect symmetry distal interphalangeal joint arthritis, one or more ankylosed joints. In approximately 5 percent of persons with psoriatic arthritis the character of joint injury becomes profoundly destructive resulting in what is sometimes referred to as arthritis mutilans.

### **Pediatric psoriatic Arthritis**

It usually resembles the disease in adults. However isolated tenosynovitis and non articular involvement are more prevalent. Occasional childhood cases stimulate a picture of Still's disease but with little constitutional involvement. Criteria have been promulgated that emphasize the presence of psoriasis, dactylitis nail pitting and positive family history.

### **Other manifestations**

In occasional patients sternomanubrial and temporomandibular joints may become involved in association with extensive typical psoriatic arthritis. Eye – involvement occur in less than one – fifth of patients conjunctivitis is well recognized in psoriatic arthritis. Cardiac and pulmonary manifestations that are more characteristic of Reiter's syndrome have occasionally been reported in individuals with pustular psoriasis.



### **Radiologic Features of Psoriatic Arthritis**

The radiologic features of psoriatic arthritis closely stimulate those of rheumatoid arthritis. The changes are asymmetric destruction of the terminal phalangeal tufts may occur (acro-osteolysis) and in the most severe form the bones completely disappear. Along with destruction new bone formation also occurs. Due to this the phalanges show playing of their bases causing 'fish-tail' deformity or more commonly 'pencil-in-cup' deformity. Bony ankylosis of the joints may occur. Osteoporosis occurs less often in psoriatic arthritis. Psoriatic arthritis has a predilection for the distal interphalangeal and proximal interphalangeal joints with relative sparing of the metacarpophalangeal and metatarsophalangeal joints.

The cervical spine shows radiographic changes more frequently than the rest of the spine. Various patterns of vertebral and paravertebral ossification may be seen. Marginal and non-marginal type appears as 'inverted comma', 'Ear drop' or as 'bag-pipe' forms.

### **Laboratory Findings;**

No currently recognized laboratory findings are diagnostic of psoriatic arthritis. The inflammation is accompanied by the elevation of various acute phase reactants that implies the release of cytokines serum complement level and ESR are increased and non-specific anaemia of chronic disease is present. The blood lymphocyte findings in psoriatic arthritis include normal levels of cells with natural killer function, in contrast to lowered levels in rheumatoid arthritis and normal level of CD4 and CD8 lineage T cells class II expression on monocytes and B cells is reported to be decreased molecules present and endogenous antigen to CD8 lineage T Cells.

# PROTOCOL

## AN OPEN TRIAL OF *SIDDHA TREATMENT (SHENKOTTAI NEI AND SIVAPPU ENNAI)* FOR *KALANJACHAGAPADAI* (PSORIASIS)

BY

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### 1. BACKGROUND

Psoriasis is a non-infectious chronic inflammatory disease of the skin characterized by well defined erythematous plaques with large, adherent, silvery scales. The main abnormality in psoriasis is increased epidermal proliferation due to excessive division of cells in the basal layers. The prevalence of psoriasis is between 1% and 3% in most populations. It may start at any age but is rare under 10 years and often seen between 15 and 40 years. The course of disease is usually chronic with exacerbations and remission.

In *Siddha Veidhya Thirattu* text, there is a preparation named “*Shenkottai Nei*” which is indicated for “*Kuttam*”. In *Siddha Maruthuvam Sirappu* text there is a external application named “*Sivappu Ennai*” which is indicated for skin disease.

Kalanchaga Padai is one among the 18 types of *Kuttam*. *Kalanchagapadai* can also be compared with *Virpodaga Kuttam*, *Teathuru Kuttam* and *Sadharu Kuttam*.

*Vetpaalai*, *Avuri* are the commonly used medicinal plants in Siddha medicines for Kalanchagapadai. *Shenkottai Nei & Sivappu Ennai* are not commonly used in practice. So, I would like to estimate their efficacy in a open clinical trial in the OPD & IPD patients at the National Institute of Siddha, Chennai.

### 2.AIMS

#### (a) Primary aim

To estimate the efficacy of “*Shenkottai Nei* and” *Sivappu Ennai*” in the treatment of psoriasis.

#### (b) Secondary aim

To find out the side-effects or adverse reactions of the drugs, if any.

### **3. POPULATION & SAMPLE**

The population consists of all patients with psoriasis satisfying the inclusion and exclusion criteria mentioned below. The sample consists of patients attending the IPD/OPD of the Ayothidoss Pandithar Hospital of the National Institute of Siddha, Chennai-47.

### **4. SAMPLE SIZE**

The trial size will be 50 patients.

### **5. INCLUSION CRITERIA**

1. Aged 13 years and above.
2. Willing to give blood specimen for the investigation when required.
3. Willing to be in-patient for 49 days, or willing to attend OPD once in 8 days for 48 days.

### **6. EXCLUSION CRITERIA**

Patients of Lichen planus, Pityriasis rosea, Diabetes Mellitus, Hypertension, Hyper cholestremia, Jaundice, heart ailments like Angina (or) any other serious illnesses are not eligible for the trial.

### **7. WITHDRAWAL CRITERIA**

1. Any drastic changes occurring in haematological parameters.
2. Development of severe exacerbation.
3. Occurrence of any other serious illness.

### **8. TRIAL DRUG & DURATION**

Shenkottai Nei	-	2 g twice a day, after food with hot water.
Sivappu Ennai	-	25 ml local application twice a day.
Trial treatment period	-	48 days.

### **9. TESTS & ASSESSMENTS**

#### **(a) Clinical assessment**

Macules, Papules, Pustules, Plaques of erythema, Candle-grease sign, Auspitz sign, Itching and Koebner's phenomenon.

## **(b) Investigations**

**Blood tests** : TC, DC, ESR, Hb, Blood Sugar, Cholesterol, Blood Urea, Serum Creatinine.

**Urine tests** : Albumin, Sugar, Deposits

**Motion tests** : Ova, Cyst, Occult blood.

## **10. CONDUCT**

Psoriasis patients satisfying inclusion and exclusion criteria will be admitted to the trial. Informed consent will be obtained from the patients.

A day before starting trial treatment, cleaning of *mukku* by purgation (Poovarasam pattai ennai) will be carried out. Photos will be taken and tests will be carried out before during treatment and at the end of the treatment.

For IP patients, the trial drug will be given by the doctor. For OP patients, the trial drugs will be given for 8 days. They will be asked to come to OP with unconsumed medicines and return them. On the 8<sup>th</sup> day, the trial drug will be given to the patient for another 8 days. At each clinic visit, clinical assessment will be taken.

## **11. FORMS**

**Form I** – Selection proforma - Used before admission of the patients to the trial.

**Form II** – Assessment proforma - Used once in 8 days during treatment.

## **12. ANALYSIS**

Changes in the proportion of patients before and after treatment for signs and symptoms will be analysed using Paired  $X^2$  – test.

## RESULTS AND OBSERVATION

**Table :1                      Gender distribution**

<b>Gender</b>	<b>Cases</b>	
	<b>No</b>	<b>Percentage or (%)</b>
Male	42	70.0
Female	18	30.0
Total	60	100.0

**Observation:**

For the study on Kalanchaga padai, 60 patients were selected of which 70% cases were males and 30% of cases were females

**Table :2                      Age distribution**

<b>Age Group (years)</b>	<b>Cases</b>	
	<b>No</b>	<b>Percentage or (%)</b>
13 - 20	7	11.7
21 – 30	14	23.3
31 – 40	14	23.3
41 – 50	15	25.0
51 – 60	9	15.0
Above 60	1	1.7
Total	60	100.0

**Observation:**

Out of 60 cases taken for clinical trial most of the cases were above 20 years

### 3. Paruvakaalam

Among 60 patients 5 (8.3%) cases were admitted to the trial in Karkaalam, 37 (61.7) cases were admitted in Koothikaalam, 18 (30%) were admitted in Munpanikaalam, No case was admitted in Pinpani, Elavenil & Muthuvenil kaalam.

### 4. Kaalam Distribution:

According to siddha literature human life can be classified into three periods, each of them having approximately 30 years age with respect to Vaatham, Piththam and Kabham doshas dominance during different age period.

**Table : 3 Kaalam distribution**

Kaalam	Cases	
	No	Percentage or (%)
Vaatha Kaalam (1-33 years)	25	41.7
Piththa Kaalam (33-66 years)	35	58.3
Kabha Kaalam (66-99 years)	0	0
Total	60	100.0

### Observation:

Out of 60 patients, 35 (58.3%) cases were found to be in Piththakaalam, 25(41.7%) cases were found to be in Vaatha kaalam.

### 5. Gunam:

Out of 60 patients, all the patients had Rajo gunam.

### 6. Thinai:

Out of 60 cases, 56 (93.3%) in Neithal nilam and 4 (6.7%) cases in Marutha Nilam.

## 7. Duration of Illness:

**Table .4          Duration of Illness**

<b>Duration of Illness (Months)</b>	<b>Cases</b>	
	<b>No</b>	<b>Percentage or (%)</b>
1 – 2	21	35.0
3 – 4	9	15.0
5 – 6	10	16.7
7 – 8	1	1.7
8 –12	6	10.0
13-24	4	6.7
25 - above	9	15.0

## 8. Mode of onset

**Table 5 :                  Mode of onset**

<b>Mode of onset</b>	<b>Cases</b>	
	<b>No</b>	<b>Percentage or (%)</b>
Acute State	3	5.0
Subacute state	4	6.7
Chronic State	53	88.3
Total	60	100.0

### **Observation:**

Out of 60 cases, 3(5%) cases in acute state, 4 (6.7%) cases in subacute state, 53 (88.3) cases in Chronic state.

## 9. Occupational Status

**Table – 6 Occupational status**

Occupation	Cases	
	No	Percentage or (%)
Painter	5	8.3
Leather Workers	4	6.7
Mason	6	10.0
Teacher	2	3.3
Traffic Police	1	1.7
Security	4	6.7
Engineer	2	3.3
Students	8	13.3
Home makers	10	16.7
Farmer	12	20.0
Driver	6	10.0
Total	60	100.0

### Observation:

Out of 60 cases 5(8.3%) cases were painters, 4(6.7%) cases were leather workers, 2(3.3%) cases were teacher, 1(1.7%) case was traffic police, 4(6.7%) cases were security, 2(3.3%) cases were engineer, 8(13.3%) cases were students, 10(16.7%) cases were home makers, 12(20%) cases were farmer, 6(10%) cases were driver. 6(10%) cases were masson.



**10. Diet habits:****Table – 7                      Diet habits**

<b>Diet Habits</b>	<b>Cases</b>	
	<b>No</b>	<b>Percentage or (%)</b>
Vegetarian	3	5.0
Non Vegetarian	57	95.0
Total	60	100.0

**Observation:**

Out of 60 cases, 3(5%) were taking vegetarian diet and 57(95%) were taking non – vegetarian.

**11. Socio Economic Status:****Table – 8                      Socio Economic Status**

<b>Socio – Economic status</b>	<b>Cases</b>	
	<b>No</b>	<b>Percentage or (%)</b>
Poor	11	18.3
Middle class	45	75.0
Rich	4	6.7
Total	60	100

**Observation:**

Out of 60 cases, 11(18.3%) of cases were poor, 45(75%) cases were middle class and 4(6.7%) cases were rich.

## 12. Clinical Features

**Table – 9 Clinical Features**

Clinical features	Cases	
	No	Percentage or (%)
Erythematous patches with white silvery scales	60	100
Itching	60	100
Auspitz Sign	58	96.7
Koebner's Phenomenon	35	58.3
Scalp Lesion	54	90.0
Nail changes	5	8.3
Palm and sole lesion	10	16.7
Painful joints	5	8.3

## 13. Three Dhosas

### a. Vaatham:

Out of 60 patients abanan were affected in 2(3.3%) cases.

Viyanan was affected in 60(100%) 60 cases

Koorman was affected in 1(1.7%) case.

### b. Piththam:

Out of 60 patients Ranjagam was affected in 60 (100%) cases.

Sathagam was affected in 10(16.7%) cases

Alosagam was affected in 1 (1.7%) cases

Prasagam was affected in 60 (100%) cases.

### c. Kabam:

Out of 60 patients Santhigam was affected in 5(8.3%) cases.

#### 14. Udal Kattukal

**Table : 10**

Udal Kattukal	Cases	
	No	Percentage or (%)
Saaram	60	100.0
Senneer	60	100.0
Oon	60	100.0
Kozhuppu	0	0
Enbu	5	8.3
Moolai	0	0
Sukkilam / Suronitham	0	0

Out of 60 patient saaram, Senneer and Oon were affected in 60(100%) cases.  
Enbu was affected in 5(8.3%) cases.

#### 15. Ennvagai Thervugal:

Out of 60 patients Niram and Sparism were affected in 60(100%) cases. Malam was affected in 2(3.3%) cases.

#### 16. Naadi:

**Table : 11 Naadi**

Naadi	Cases	
	No	Percentage or (%)
Vaatham	0	0
Piththam	0	0
Kabam	0	0
Vaatha Piththam	43	71.7
Vaatha Kabam	1	1.7
Piththa Kabam	2	3.3
Piththa Vaatham	5	8.3
Kaba Vaatham	4	6.7
Kaba Piththam	5	8.3
Total	60	100.0

### 17. Neerkuri & Neikuri

Table : 12

Type of Test	Cases	
	No	Percentage or (%)
Neerkuri 'Vaikkol Niram'	60	100
Neikuri Spreading pattern		
a. Aravena Neendathu	0	0
b. Azhipol paraviathu	0	0
c. Muthuthoththu Nitral	60	100

### 18. Ganaenthiriyam:

Out of 60 patients mei was affected in 60(100%) cases.

### 19. Kanmendhirium

Table : 13

Kanmendhirium	Cases	
	No	Percentage or (%)
Kai	10	16.7
Kaal	15	25
Vaai	0	0
Eruvaai	2	3.3
Karuvaai	0	0

#### Observation:

Out of 60 patients kai was affected in 10(16.7%) cases. Kal was affected in 15(25%) cases. Eruvai is affected in 2(3.3%) cases.

## 20. Precipitating Factors:

**Table 14**

Precipitating Factors	Cases	
	No	Percentage or (%)
Climate		
a. Winter	4	6.7
b. Summer	25	41.7
c. Winter & Summer	15	25
Occupation related	15	25.0
Anxiety	14	23.3
Chloroquin treatment of chikungunya fever	4	6.7
Hereditary	2	3.3
Physical Injury	2	3.3

## 21. Clinical Features After Treatment

**Table : 14**

Clinical features	Before treatment				After treatment			
	No	Mild	Moderate	Severe	No	Mild	Moderate	Severe
Itching	0	0	23	37	60	0	0	0
Bleeding	2	0	39	19	60	0	0	0
	Present		Absent		No	Reduced	Persistent	Aggravated
Erythema	60		0		53	7	0	0
Scaling	60		0		57	3	0	0
Pustule	0		60		60	0	0	0
Papule	1		59		1	60	0	0
Macule	60		0		52	8	0	0
	No		Hypo	Hyper	No		Hypo	Hyper
Pigmentation	0		0	60	8		2	50

## 22. Result of Treatment

Table 15.

Result of Treatment	Cases	
	No	Percentage or (%)
Good improvement	51	85
Moderate improvement	9	15
Mild Improvement	0	0
No improvement	0	0
Total	60	100



Table No.    **Results of Statistical Analysis Objective parameters observed Before  
and After Treatment of 60(n) patients of Kalanjagapadai,  
National Institute of Siddha, Chennai – 600 047, During 2007-08**

	Parameter	Mean			Paired t-test	Probability (P) Value	Statistical Significance of the Difference
		Before R <sub>x</sub>	After R <sub>x</sub>	Difference			
1.	Urea (mg%)	26.9	25.8	1.1	t = 1.668	P>0.05	Not Significant
2.	Creatinine (mg%)	0.95	0.80	0.15	T=2.007	P<0.05	Significant
3.							



## DISCUSSION

Kalanchaga Padai is one of the commonly encountering skin disease in a day – today dermatological practice. Eventhough it is a condition, which produces various mental harasments and annoyance to affected individuals. Moreover, everyone wants to their physique to attractive, but this disease makes the skin silvery scaly apperance and the peal of skin in some what so apparent and obvious.

Perhaps Kalanchaga padai disease is not contagious. The affected individuals may be deprived from the society. Relapse and remissions of this disease is quite common and there may be no specific treatment in the other systems of medicine but siddha system enfarages cure to this disease. Any attempt made to evaluate the nature of the disease leading to the way of treatment is itself an achievement. In this way the present attempt is considered a mile stone.

For this dissertation study, 60 patients were selected and 20 patients were admitted in National Institute of Siddha, Sirappu Maruthuvam Department as in – patient and 40 patients were treated as Out patient department of Sirappu Maruthuvam Department with the trial drug.

The trial drugs are very useful in skin diseases as mentioned in Siddha literature were selected and trial drug was prepared as per the text.

The biochemical, pharmacological and toxicoligical study of the trial drug were tested in the laboratories of Dr. C.L. Baid Mehta College of Pharmacy, Thoraipakkam, Chennai. The results were documented and interpreted for the Prognosis of the disease.

Based on the various criterias, the datas were collected and tabulated. The criterias were the Sex predominance, incidence of the disease with respect to the kaalam (Life Span), seasonal variation, clinical manifestation, relation to occupation, diet and the assessment of the improvement in the prognosis of the disease with the trial drug.

The clinical study revealed that the incidence of the disease was highest in Piththa kaalam (33066 yrs.) 58.3%.

In seven udar kattugal 100% of cases had derangement in Saaram, Seener and Oon. Affected Saaram produced depression. Affected Seneer produced erythematous lesions and impurities in the blood for example anaemia raised ESR.

In Envagai thervugal, The skin lesions of the Kalachaga Padai colour (Niram) and sense of touch (sparism) were affected in all the 60 cases, there was dryness of the skin, roughness, thickness and silvery scaly appearance of the skin were found out.

Out of 60 cases, 100% had Rajo gunam. From this inference ones character is very much important in developing disease. This was clearly stated in the siddha system of medicine. So control of mind and restoration of normal life style can lead to reduction in the formulation of disease.

88% of cases were presented their complaints in the chronic state. This shows that majority of patients did not come in the early stages, because of their ignorance and negligence of all the diseases. Moreover if there was no complete remedy in the other system of medicine then they tried with siddha medicine.

Laboratory investigations of Blood, Urine, Neerkuri, Neikuri and Stools were done for all 60 cases.

All the patients were instructed to following pathiyam.

After discharge of the cases all the patients were advised to attend out patients department of Sirappu Maruthuvam for further followup.

During the time of discharge the patients were uniformly advised to follow Yogasanas and Pranayamam. These methods helped them as a supportive therapy of the patients felt better in practising them.

Blood, Urine and Motion analysis were once again done after the completion of treatment. The increased ESR were noted to be normal.

## SUMMARY

The diseases Kalanchaga Padai was taken for the clinical study. The clinical study on Kalanchaga padai with reference to its aetiology, pathogenesis, investigations, clinical features, diagnosis and treatment were conducted at the post graduate department of Srappu Maruthuvam, National Institute of Siddha, Tambaram Sanatorium, Chennai-47. Sixty cases with the signs and symptoms of Kalanchaga Padai were selected and a thorough observation was made. They were gathered from the out patient section and admitted in the Inpatient ward for the followings.

No drug reactions like nausea, vomiting, diarrhoea, abdominal discomfort, skin rashes, drowsiness were reported during the study period. The drugs administered in the clinical study were used only after careful purification process laid down for them individually.

The majority of the patients were male. The trial drug have mainly Kaippu suvai and this mainly accounts for the therapeutic effect in skin diseases.

The results were found to be encouraging. Among the in patient treated 80% showed good results. 20% showed moderate results. Along with the in – patients, 40 patients were treated under the out patient ward to 34 cases showed good result. 6 cases showed moderate results.

## CONCLUSION

“உற்றானளவும் பிணியளவும் காலமும்

கற்றான் கருதிச் செயல்”

-- திருக்குறள்

According Tiruvalluvar in his Thirukkural, stated that before designing a treatment, we should look into the Physical condition of patient, condition of disease, duration and onset of disease. Treatment was given for the Kalanchaga padai on the basis of “Mukkuutra Theory”. The deranged kuttrams were corrected by the medicine given ‘**Shenkottai nei**’ and ‘**Sivappu ennai**’ as an internal and external respectively. The above medicines were selected from the **siddha literturs Siddha Veidhiya Thirattu and Siddha Maruthuvam Sirappu** respectively. Almost all the cases treated with above medicines were shown remarkable improvement.

No adverse effects were reported during the course of treatment and the drug evaluation was done with the modern parameters.

A length it is concluded that in the treatment of Kalanchaga Padai with **Shenkottai nei** and **Sivappu ennai** were found to be very safe and also economical.

# PROPERTIES OF TRIAL MEDICINE

## சேங்கொட்டை நெய் (Internal Medicine)

ஆதாரம் : சித்தவைத்தியத் திரட்டு, பக்கம் 256, முதல் பதிப்பு பிப்ரவரி 1998

### Ingredients:

சுத்தித்த சேங்கொட்டை	- 5 பலம் (175 கிராம்)
பசுவின் பால்	- 1 படி (1.3 லிட்டர்)
பசுவின் நெய்	- தேவையான அளவு

### செய்முறை

சுத்தித்த சேங்கொட்டையை சிறு துண்டுகளாக வெட்டி உட்பருப்பை நீக்கி பசுவின் பாலை மூன்று பங்காக்கி ஒரு பங்கில் கொட்டையிட்டு காய்ச்சி எடுத்து மற்ற இருபங்கிலும் தனித்தனி இவ்விதம் காய்ச்சி எடுத்த பாலுக்கு சமளடை பசுவின் நெய் சேர்த்து நெய் பதம் வரும் வரை காய்ச்சி எடுக்க வேண்டும்.

அளவு : 2 கிராம். இரு வேளை.

தீரும் நோய்கள் : தோல் சம்மந்தமான பற்பல நோய்கள், காசம், சுவாசம், கயம் இவைகளும் நீங்கி உடல் பலம் உண்டாகும்.

### சேங்கொட்டை

**Botanical Name** : *Semecarpus anacardium*. Linn

**Family Name** : Anacardiaceae

வேறுபெயர்கள் : சேராங்கொட்டை, வல்லாதி, வல்லாதகி, எரிமுகி, பல்லாதகி கிட்டாக்கனிகொட்டை, நந்திவித்து

பயன்படும் உறுப்பு : கொட்டை பருப்பு

சுவை : கைப்பு - விறுவிறுப்பு தன்மை - வெப்பம் பிரிவு - கார்ப்பு

### செய்கை:

உடற்றேற்றி - Alterative

புண்ணாக்கி - Caustic

### சுத்தி:

சேங்கொட்டையின் மேல் மரம் போல் உள்ளமூக்கைச் சீவி புளி இலை குடிநீரிலும், பசுஞ்சாணிப்பாலிலும் கற்றாழைச்சாற்றிலும் முறையே வேகவைத்து எடுக்க வேண்டும்.

### பொதுகுணம்:

குட்டங் கயரோகங் கொல்லும் விடபாகந்  
துட்டந் தருகிருமி சூலையும் போம் - மட்டலருங்  
கூந்தன்மயி லேகிரந்திக் கூட்டம்போஞ் செங்கையில்  
ஏந்துசேங் கொட்டைதனை யே.

சேங்கொட்டை மெயத்திமிரைத் தீராக் கடிவிடத்தைப்  
பாங்கொட்டு மூலத்தைப் பற்றறுக்கும் - ஆங்கெட்டிக்  
கொல்லும்வா தத்தினொடு குன்மத்தை யும்மதனை  
வெல்லும் அயிற்கண்ணாய் விள்.

- அகத்தியர் குணவாகடம்

இது பெரு நோய், இளைப்பு நோய், நஞ்சுகள், சூலை இவைகளைப்  
போக்கடிக்கும். மேலும் திமிர்ப்படை, கருப்புப்படை, வெண்படை (வெண்குட்டம்),  
தீராக்கடி, மூலம், வளிநோய்கள், குன்மம் இவைகளையும் விலக்கும்.

இ.தன்றியும் இது சொறி, சிரங்கு, புண், கீல், பிடிப்பு, சூலை முதலிய  
நோய்களையும் போக்கவல்லது.

Shenkottai fruits used for ascites, rheumatism, asthma, neuralgia, epilepsy and  
psoriasis and also for warts and tumours. Biological tests have shown that extract of the  
fruit is effective against human epidermoid carcinoma of the nasopharynx in tissue  
culture: extract also shows hypoglycaemic action.

### **Chemical Constituents :**

Kernel of the nut contains a small quantity of sweet oil; “the pericarp of the fruit  
contains a bitter and powerful astringent principle (which is universally used in India as a  
substitute for marking ink). The black corrosive juice of the pericarp contains a tarry oil  
consisting of 90 per cent of an oxy – acid named anacardic acid and 10 per cent of a  
higher, non volatile alcohol called cardol. Naidu (1925) isolated catechol and a mono –  
hydroxyphenol which he called ‘anacardol’, besides two acids and a fixed oil from the  
kernel of the nut”. Pericarp also contains a vesicating oil 32 p.c., soluble in ether and  
which blackens on exposure to the air. Fruit yields 2.14 p.c. of ash.

Chloroform extract of nuts significantly increased life – span in ascites tumor  
systems and solid tumor systems (B-16 melanoma and Glioma – 26)

Isolation of a biflavonoid – jeediflavanone – from nutshells another biflavonoid – galluflavaone – from nutshells a third biflavanoid – semecarpuflavanone – from nutshells: a new dimeric flavonoid – nallaflavaone – isolated and its structure determined and confirmed: isolation of another new biflavonoid – semecarpetin – from nutshells and its characterisation.

பால்

**Lactus**

வேறுபெயர் : பயம், சீரம், சுதை, பயசு, பாகு, அழுது, துத்தம், சாறு

**பொதுகுணம்:**

”பாலர் கிழவர்பழஞ்சுரத்தோர் புண்ணாளி  
சூலையர் மேகத்தோர் துரபலத்தோர் ஏலமிவர்  
எல்லார்க்கும் மாகும் இளைத்தவர்க்குஞ் சாதகமாய்  
நல்லாய் பசுவின்பால் நாட்டு”

”உணவின் பகுதியை யோதறுந் றனமினிப்  
பணிவுள வெண்பசும் பாலினைப் பாற்று  
நற்பசு வின்சுதை நாவினைப் பாற்றிடும்  
பொற்பான செம்பசு போக்குங் கயப்பிணி  
புள்ளியா மேகநோய் போக்கிடு நிச்சய  
மடாச்சுதை தீபனமாக்கிடு மட்டபால்  
விடாத்தொடர் காத்திர வெம்மையை மாற்றுமே”

**Chemical Constituents**

Fat – 3.5%	Casein	Zinc	Vitamin (Vit –A)
Sugar – 4.5%	Calcium	Lactose	
Lactoalbumin – 0.7%	Phosphorus	Sodium	

பசுவின் நெய்

**Cow's Ghee**

தாக முழலைசுட்குஞ் சர்த்திபித்தம் வாயுபிர

மேகம் வயிற்றெரிவு விக்கலழல் - மாகாசங்

குன்மம் வறட்சி குடற்புரட்ட லஸ்திசுட்கஞ்

சொன்மூலம் போக்குநிறைத் துப்பு

பசுவினது நெய்யானது தாகம், உழலைப் பிணி, அதிசுட்கரோகம், வாந்தி, பித்தாதிக்கம், வாதவிஷமம், விரணப்பிரமேகம், வயிற்றிலெரிவு, பித்தவிக்கல், இருமல், வயிற்றுவலி, சினைப்பு, குடல் நெளிதல் , எலும்புரக்கி பல மூலரோகம் ஆகிய இவைகளை நீக்கும்.

### சிவப்பு எண்ணெய் (External Medicine)

ஆதாரம் : சித்த மருத்துவம் சிறப்பு பக்கம் - 290

டாக்டர். ஆர். தியாகராஜன் L.I.M

#### Ingredients

புங்கன் வேர்	-	4 கிலோ
தேங்காய் நெய்	-	1 கிலோ
மஞ்சிட்டி	-	62 கிராம்
நன்னாரி	-	62 கிராம்
தேன்மெழுகு	-	62 கிராம்
வெள்ளைக் குங்கிலம்	-	52 கிராம்
சுருள் பட்டை	0	3 கிராம்
செவ்வல்லிக்கொடி	0	10 கிராம்

#### செய்முறை:

புங்கன் வேர் மேல் பட்டையையச் சீவி பால் எடுத்து அதில் தேங்காய் எண்ணெய் கலந்து அடுப்பேற்றி அதில் மஞ்சிட்டி, நன்னாரி இவ்விரண்டையும் இடித்து பாதி சரக்கை கியாடி மிட்டு மீதியைச் சூரணித்துக் கற்கமாக்கி சேர்க்க வேண்டும். கொதித்து பாகம் வரும் சமயத்தில் சுருள் பட்டை, செவ்வல்லிக் கொடி இரண்டையும் இடித்து சூரணித்து வடிகட்டும் தறுவாயில் கலந்து வடிகட்டும் பொழுது தேன் மெழுகு, வெள்ளைக் குங்கிலியம் இவ்விரண்டையும் ஒரு பாத்திரத்திலிட்டு அதில் மேற்கண்ட தைலத்தை வடிகட்ட வேண்டும்.

தீரும் நோய்கள் : கரப்பான், தோல் நோய்கள்

### Description of Drugs

#### புங்கன்

Botanical Name : Pongamia Glabra



Family : Papilionaceae

வேறுபெயர்கள்

புன்கு, பூந்தி, கரஞ்சகம், கரஞ்சம்

பயன்படும் உறுப்பு : இலை, பூ, விதை, வேர் எண்ணெய்

சுவை : கைப்பு, துவர்ப்பு, தன்மை - வெப்பம், பிரிவு - கார்ப்பு

செய்கை

பொது

துவர்ப்பி - Astringent

உடற்றேற்றி - Alterative

தூக்குணிப்புழுவகற்றி - Parasiticide

எண்ணெய்

அழுகலகற்றி - Antiseptic

வெப்பமுண்டாக்கி - Stimulant

**பொதுகுணம்:**

”புண்ணும் புரையுமறும் பொல்லாத வாதமறும்  
கண்ணும் கிரந்திபடை நாசமுறும் - மண்ணுலகில்  
தங்கம்போல் மேனி தழைத்தோங்கு மெந்நாளும்  
புங்கம்பாற் செய்தி புகல்”

புங்கம் பாலால் புண், புரையோடும் ரணம், வாயு, கிரந்தி, படை இவைகள் போம்.  
இன்னும் தேகம் நல்ல மேனியை அடையும்.

**தேங்காய் நெய்**

Botanical Name : Cocos Nucifera

Family : Palmae

**பொதுக்குணம்:**

”தேங்காயி னெய்யதனாற் றியால் வருபுண்போம்

பாங்காகக் கூந்தற் படர்ந்தேறு - நீங்காத  
பல்லடியின் னோயும் படர்தா மரை சிரங்கும்  
அல்லறப் போமென் றறி

தேங்காய் நெய்யால் தீப்பட்ட புண்ணும், பல்நோய், படர்தாமரை, சிரங்கு  
ஆகியவைகள் போம். தலைமயிர் வளரும்

#### Chemical Constituents:

Cocoonut oil contains free caprylic acid in addition to glycerides of lauric, myristic, palmitic and stearic acids.

Coconut contains 92% saturated fat, 6.5% mono saturated fat, 1.5% polyunsaturated.

Coconut oil contains a type of saturated fat called medium chain triglycerides (MCTS). These MCTS have immune supporting properties, especially lauric acid, a saturated fatty acid that is found in significant amounts in coconut oil.

#### மஞ்சிட்டி

Botanical Name : Rubia cordifolia

Family : Rubiaceae

வேறுபெயர் : பாண்டி, மஞ்சூகம், மஞ்சிட்டி

பயன்படும் உறுப்பு : வேர்

சுவை : கார்ப்பு, கைப்பு, தன்மை - வெப்பம், பிரிவு - கார்ப்பு

#### செய்கை:

சூதகமுண்டாக்கி - Emmenagogue

துவர்ப்பி - Astringent

சிறுநீர்ப்பெருக்கி - Diuretic

#### குணம்:

இது சூதக அழுக்கை நீக்கும். குருதி நீர் முதலியவைகளுக்குச் செந்நிறத்தை ஊட்டும்.

A paste made by rubbing up the roots with honey is a valuable application for freckles and other discolouration of the skin; also in external inflammations, ulcers and skin diseases such as pityriasis versicolor, etc.

## Chemical Constituents:

Roots contain resinous and extractive matter, gum, sugar. Colouring matter and salts of lime. Colouring matter consists of a red crystalline principle – purpurin: a yellow principle glucoside – manjistin, garancin and xanthine (Yellow)

### தேன்மெழுகு

### CERA, WAX

செய்கை : உள்ளழலாற்றி

#### பொதுகுணம்:

”அறைபக்க வாத மதைப் பைய மூதை  
குறைவிந்தி தழ்நோய் தேள்கூளி - கறையைப்  
புழுகெடுக்க வங்கமுறு புண்ணிடிப்புண் டிப்புண்  
மெழுகெடுக்க வாங்கலு மெய்”.

தேன் மெழுகினால் பாரிச வாயு, வீக்கம், கபநோய், வாதகோபம், சுக்கில நஷ்டம், சிலேஷமோஷட ரோகம், தேள்விடம், பைசாசம், குஷ்டம், ஆகந்துக விரணம் சந்தியோ விரணம் முதலியன நீங்கும்.

### வெள்ளைக் குங்கிலம்

Botanical Name : Shorea Robusta

Family : Dipterocarpaceae

வேறுபெயர் : சருவரசம்

சுவை : கைப்பு, தன்மை : வெப்பம், பிரிவு : கார்ப்பு

#### செய்கை

வெப்பமுண்டாக்கி - Stimulant  
கோழையகற்றி - Expectorant  
சிறுநீர்ப்பெருக்கி - Diuretic

#### பொதுகுணம்:

”வெள்ளை யளித்த விரணநா பிக்கமலத்  
தொள்ளைவிர ணம்கேந் தோற்றுகினும் - உள்ளே

வருவரசனை மேற்புண் வரினுஞ் சுவேதச்  
சருவரச மேற்பழியைச் சாற்று”

வெள்ளைக்குங்கிலியத்தால் வெட்டையால் பிறந்த நீர் நாளப் புண், தொப்பூழ்ப்புண்,  
சீழ் மேகம், உள்மூலப்புண் ஆகியவை நீங்கும்

#### சுருள்பட்டை

Botanical Name : Capparis Aphylla.

Family Name : Capparideae

பொதுகுணம் :

“செங்கத்தா ரிச்சடைக்குத் தீராத வன்மேகம்  
பொங்கி வருங்கிரந்திப் புண்புரைகள் - தங்குகின்ற  
சந்திகசி லேஸ்மந் தனித்த மகாவாத  
முந்திவிட் டோடு மொழி”

செங்கத்தாரிவேர் பட்டையால் பிரமேக நீர் ஒட்டுப் புண், புரைகள், கீல்களில்  
வீங்கச் செய்கின்ற கபமகாவாதம் இவைகளைப் போக்கும்.

#### செவ்வல்லிக்கொடி

Botanical Name : Nymphaea Stllata

Family Name : Nymphaeaceae

வேறுபெயர் : ஆல்பம், குமுதம், கைரவம்

பயன்படும் உறுப்பு : பூ, கொடி, இலை, கிழங்கு, காய்

சுவை : சிறு துவர்ப்பு தன்மை : தட்பம் பிரிவு : இனிப்பு

செய்கை: வறட்சியகற்றி - Emollient

சிறுநீர்ப்பெருக்கி - Diuretic

பொதுகுணம்:

செவ்வள்ளி யின்கொடிக்குச் சேடித்த குட்டமொடு  
கவ்வு குறைநோய் கரப்பானு - மிவ்வுலகின்  
மானே குடல்வாதம் வல்ல படுவனுட  
னானபரு மூலமும்போ மால்”

செவ்வள்ளிக் கொடிக்கு வீறு குட்டம், பெருவியாதி, கடுவன், குடலண்டம், நகம்,  
குதிகால்களைப்பற்றிய கொப்புளம், மூலம் இவைகள் தீரும்.

#### நன்னாரி

Botanical Name : Hemidesmus Indicus  
 Family Name : Asclepiadaceae  
 வேறுபெயர் : அங்காரி மூலி, நறுநெட்டி பாதாளமூலி,  
 கோபாகு, சாரிபம், பாற்கொடி, நீறுண்டி,  
 கானனுசாரி, கிருஷ்ணவல்லி, சாரியம்  
 பயன்படும் உறுப்பு : வேர்  
 சுவை - இனிப்பு, சிறுகைப்பு, தன்மை - தட்பம், பிரிவு - இனிப்பு

### செய்கை

உடற்றேற்றி	Alterative
உரமாக்கி	Tonic
உள்ளழலாற்றி	Demulcent
சிறுநீர்ப்பெருக்கி	Diuretic
வியர்வைப் பெருக்கி	Diaphoretic

### பொதுக்குணம்:

நன்னாரி வேரை நறுக்கி யரைத்துமதன்  
 வின்னாரி யூனால் வெதுப்பியே - தின்னவெறி  
 தோணாது கற்றாழைச் சோற்றிற் கலந்துண்ணக்  
 காணாது வண்டு கடி”  
 - பதார்த்தகுண விளக்கம்

சலதோடம் பித்தமதி தாகம் உழலை  
 சலமேறு சீதமின்னார் தஞ்சு - சூலக மதிற்  
 சொன்னமது மேகம் புண்சுரமுவையெ லாமொழிக்கும்  
 மென்மதுர நன்னாரி வேர்  
 - தேரன் குணவாகடம்  
 அங்கா ரிகைமூலி யாச்சியத்தோ டுண்ணா நித்திட  
 யங்கா ரிகைமூலி யாளுமே - யங்காரி  
 பற்றாது  
 - தேரன் யமக வெண்பா

நன்னாரிவேரை அரைத்துத் தேனில் மத்தித்து உண்ணில் அதிபித்தம் தீரும். அதைக் கற்றாழைச் சாற்றுடன் உண்ண வண்டுகடி போம். இன்னும் கிரமப்படி உபகோகிக்க நீரேற்றம், பித்தநோய், தாகம், வாய்நீர்ச்சுரப்பு, புணர்ச்சியின் சூடு, மதுநீர், கிரந்தி, சுரவேகம் இவைகள் போம் என்க

### **Chemical Constituents:**

Coumarin (the aroma and taste of the drug are due to this constituent), a volatile oil. A crystallizable principle – hemidesmine, and a crystalline stearoptin called smilasperic acid. “Recent researches by Allopaths have proved conclusively that the active principles of Sarsaparilla consist of an enzyme, an essential oil and a saponin.

## **PRECLINICAL STUDIES**

### **BIO CHEMICAL ANALYSIS**

#### **QUALITATIVE ANALYSIS OF ACIDIC/BASIC RADICALS AND PHYTOCHEMICAL CONSTITUENTS IN TEST DRUGS**

<b>Procedure</b>	<b>Observation</b>	<b>inference</b>
<b>Test for Calcium:</b> 2 ml of extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxide solution.	White precipitate is not formed	Absence of calcium
<b>Test for Sulphate :</b>	White Precipitate is not	Absence of Sulphate

2 ml of the extract is added to 5 % barium chloride solution.	formed	
<b>Test for Chloride :</b> The extract is treated with Silver nitrate solution	White precipitate is formed	Present of Chloride
<b>Test for carbonate :</b> The substance is treated with Conc. HCl.	Effervescence is not formed	Absence of carbonate
<b>Test for Starch :</b> The extract is added with weak iodine solution	Blue colour is not formed	Absence of starch
<b>Test for Iron (Ferric) :</b> The extract is treated with glacial acetic acid and potassium ferrocyanide	Blue colour is not formed	Absence of Ferric iron
<b>Test for Iron (Ferrous) :</b> The extract is treated with Conc. HNO <sub>3</sub> and ammonium thiocyanate	Blood red colour is formed	Presence of Ferrous iron
<b>Test for phosphate :</b> The extract is treated with ammonium molybdate and conc. HNO <sub>3</sub>	Yellow precipitate is not formed	Absence of phosphate
<b>Test for Tannic acid :</b> The extract is treated with Ferric chloride	Blue black precipitate is formed	Presence of Tannic acid
<b>Test for Unsaturation :</b> 1 ml of Potassium permanganate solution is added to the extract.	Does not get decolourised	Absence of unsaturated compound
<b>Test for saponins:</b> Dilute extract+ 1ml of distilled water shake well.	Froth is formed	Presence of saponins
<b>Test for sugars :</b> <b>Benedict method ;</b> 5ml of Benedict solution heated gently then add 8 drops of diluted extract then heated in a boiling water bath.	Colour change	Indicates the Presence of sugar
<b>Test for steroids :</b> Lieberman Burchard test ; Dilute extract +2 ml acetic anhydride+conc.H <sub>2</sub> SO <sub>4</sub> .	Red colour is not formed	Absence of steroids

<b>Test for amino acids:</b> Dilute extract +2ml of Ninhydrin's soln .	Violet colour is formed	Presence of amino acids
<b>Test for proteins:</b> Biuret method ; 1ml of dilute extract+1ml of 5% CuSO <sub>4</sub> + 1% NaOH.	Violet colour is formed	Presence of Proteins
<b>Test for Flavanoids :</b> Dilute extract+ mg bits+2drops of conc.HCl and gently heated.	No formation of pink colour	Absence of Flavanoids
<b>Test for phenol;</b> Dilute extract+2drops of FeCl <sub>3</sub> soln.	Deep green colour is not formed	Absence of phenols
<b>Test for Tannins;</b> dilute extract +2ml of 10% lead acetate add.	White precipitate not formed	Absence of tannins
<b>Test for alkaloids;</b> Mayer's method; 1ml of dilute extract + 1ml reagent.	Cream colour precipitate is formed	Presence of alkaloids
<b>Test for Cardiac Glycosides (Keller Killani test):</b> 5 ml of each extracts was treated with 2ml of glacial acetic acid containing one drop of ferric chloride solution. This was underplayed with 1 ml of concentrated sulphuric acid	A brown ring formed and a violet ring formed below the brown ring	Presence of cardiac glycosides.

## PHARMACOLOGICAL STUDY

### MATERIALS AND METHODS

#### Test Drugs



The following drug was used in the study were collected and processed by the methods prescribed in standard text books of siddha medicines.

Shenkottai Nei (SN)

Shenkottai Nei prepared by the method described in (Siddha veidhiya thirattu, page No. 256)

### **Preparation of drug for dosing**

All drugs used for the study was suspended each time with 1% (w/v) solution of sodium carboxy methyl cellulose before administration.

### **Drugs and chemicals**

Alloxan monohydrate and fine chemicals used in these experiments were obtained from Sigma Chemicals company, U.S.A. Other analytical grade chemicals were obtained from S.d. Fine Chemicals Ltd., Mumbai.

### **Experimental animals**

Colony inbred animals strains of wistar rats of either sex weighing 200 0 250 g were used for the pharmacological and toxicological studies. The animals were kept under standard conditions 12:12 (day/night cycles) at 22<sup>0</sup>C room temperature, in polypropylene cages. The animals were fed on standard pelleted diet (Hindustan Lever Pvt Ltd., Bangalore) and tap water *ad libitum*. The animals were housed for one week in polypropylene cages prior to the experiments to acclimatize to laboratory conditions. The experimental protocol was approved by the Institutional Animal Ethical Committee (IAEC).

## **ANTI INFLAMMATORY ACTIVITY**

Anti inflammatory activity of SSC was evaluated in both acute and chronic models of inflammation.

### **Acute model**

#### **a. Carrageenan induced hind paw edema**

The carrageenan assay procedure was carried out according to the method of Wintar *et al.* (1962). Edema was induced by injecting 0.1 ml of a 1% solution of carrageenan in saline into the plantar aponeurosis of the left hind paw of the rats. The extracts, reference drug and the control vehicle (distilled water) were administered 60 min prior to the injection of the carrageenan. The volumes of edema of the injected and contra lateral paws were measured at +1, 3 and 5 hrs after induction of inflammation using a plethysmometer (Bhatt *et al.*, 1977) and percentage of anti-inflammatory activity was calculated.

### **Chronic model**

#### **b. Cotton pellet granuloma**

Sterile cotton pellets (weighing  $10 \pm 2$  mg) were implanted subcutaneously along the flanks of axillae and groins of wistar albino rats (Swingle and Shideman *et al.*, 1972). The extracts, reference drug and the control vehicle (distilled water) were administered as per protocol to rats everyday for a period of 7 days. On day + 8 the rats were sacrificed by cervical decapitation and cotton pellets were removed surgically, freed from extraneous tissue and weighed immediately for wet weight. One half of the pellets were dried in an incubator at 60°C until a constant weight was obtained.

### **Anti inflammatory activity of (SN) in Carrageenan induced hind paw edema in rats**

Groups	Paw volume (ml) by Mercury Displacement at Regular interval of Time				
	0min	30min	60min	120min	240min
Control	0.881±0.038	1.0795±0.132	2.231±0.078	2.245±0.077	2.28±0.059
Test	0.885±0.024	1.273±0.081	1.688±0.195	1.93±0.111	1.375±0.197
Standard (Dic.Sodium 5 mg/kg/po)	0.883±0.063 <sup>ns</sup>	0.996±0.067 <sup>**</sup>	1.02±0.064 <sup>**</sup>	0.926±0.041 <sup>**</sup>	0.896±0.026 <sup>**</sup>

n=6; Values are expressed as mean ± S.D followed by One Way ANOVA –Dunnett's multiple comparison test.

ns 0 Non significant as compared with control;

P<0.01 (\*\*) as compared with control.

#### Anti inflammatory activity of (SN) in Cotton Pellet Granuloma

Groups	Cotton pellet Granuloma method
	Dry Weight (mg)
Control	115.87 ± 15.42
Test	78.13 ± 10.59 <sup>**</sup>
Standard (Dic.Sodium 5 mg/kg/po)	70.00 ± 7.42 <sup>**</sup>

n=6; Values are expressed as mean ± S.D followed by One Way Anova using Dunnett's Test

\*\*\*P<0.01 as compared with that of control.

### ANTIPYRETIC ACTIVITY

Rats selected for the study were fasted overnight allowing water *ad libitum*. Initial rectal temperature was recorded using Hick's clinical thermometer. Pyrexia was induced by subcutaneous injection of TAB vaccine 1 ml/kg body weight. Six hrs later pyrexia was assessed and those animals that did not show a minimum rise of 1.5°C were rejected. The animals thus found fit for the study were divided into 6 groups as described above and drugs were administered. Pyrexia was recorded at hourly intervals for 3 hrs after drug administration.

#### Antipyretic activity of (SN) using Digital Rectal Thermometer

Groups	Rectal temperature (°C)				
	0 min	30 min	60 min	120 min	240 min
Control	35.90±1.18	37.23±1.24	38.27±0.34	37.20±1.08	36.46±0.88
Test	35.35±0.46	37.08±0.88	36.43±0.73 <sup>**</sup>	35.92±0.66 <sup>*</sup>	35.50±0.34 <sup>*</sup>
Standard (Dic.Sodium 5 mg/kg/po)	35.80±0.97	36.96±0.95	35.87±0.65 <sup>***</sup>	35.65±0.60 <sup>*</sup>	35.42±0.52 <sup>*</sup>

n=6, Values are expressed as mean ± S.D using Student's paired 't' test.  
P<0.05 as compared with that of control.

#### ACUTE ORAL TOXICITY STUDY

Acute oral toxicity was conducted as per the OECD guidelines (Organization of Economic Cooperation and Development) 423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 animals of a single sex per step. Depending on the mortality and /or moribund status of the animals, on the average 204 steps may be necessary to allow judgment on the acute toxicity of the test substance. This procedure results in the use of a minimal number of animals while allowing for acceptable data based scientific conclusion.

The method uses defined doses (5, 50, 300, 2000 mg/kg body weight) and the results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which cause acute toxicity

Wistar albino rats of either sex weighing 2000±250 g were fasted overnight, but allowed water *ad libitum*. Since the formulation is relatively non toxic in clinical practice the highest dose of 2000 mg/kg/p.o (as per OECD guidelines “Unclassified”) was used in the acute toxicity study.

The animals were observed closely for behavioural toxicity, if any by using FOB (Functional observation battery).

### **Repeated oral toxicity study**

Repeated oral toxicity studies can be used to get additional information regarding the toxicity profile of a chemical. Repeated oral toxicity studies are defined as those studies where the chemical is administered to the animal for a period covering approximately 10% of the expected life of the animal. Usually, the dose levels are lower than for acute studies and allow chemicals to accumulate in the body before lethality occurs, if the chemical possess this ability.

### **Experimental procedure**

The following experimental procedure was followed to evaluate the repeated oral toxicity study of

*1. Shenkottai Nei (SN)*

Group I : Control animals received 1% tween 20, 2 ml/kg/p.o. for 21 days

Group II : Aqueous extract of Shenkottai Nei (SN) at the dose Level of 500 mg/kg/p.o. for 21 days

Body weight, food intake and water intake was recorded at two intervals with simultaneous observation for toxic manifestation and mortality, if any. At the end of 21 days treatment all the animals were sacrificed by over dosage of ether anaesthesia. Blood was collected and used for haematological studies. Section of liver, kidney, and heart were dissected out and kept in 10% formalin for histopathological studies.

**Biochemical studies**

***Estimation of glucose***

Glucose was estimated using commercial Glucose estimation kit (Span Diagnostics) by the method of Barham *et al.*, (1972) and Tenscher. *et al.*, (1971).

**Aspartate aminotransferase (AST)**

Aspartate aminotransferase was estimated using commercial AST kit (Span Diagnostics) by the method of Reitman and Frankel (1957).

**Alanine aminotransferase (ALT)**

Alanine aminotransferase was estimated using commercial AST kit (Span Diagnostics) by the method of Reitman and Frankel (1957).

#### **Alkaline phosphatase (ALP)**

Alkaline phosphatase was assayed using commercial ALP kit (Span Diagnostics) by the method of King (1934).

#### **Urea**

Urea was assayed using the commercial kit (Span Diagnostics) by the method of Coulambe *et al.*, (1965).

### **1.8 Haematological studies**

#### **Erythrocyte count**

Erythrocyte count was estimated by Hemocytometer method of Ghai (1995).

#### **Total Leukocyte Count (WBC)**

Total Leukocyte Count was estimated by Hemocytometer method of John (1972).

#### **Haemoglobin**

Haemoglobin was estimated by method of Ghai (1995).

**Effect of Siddha Formulations (SN) on Haematological parameters after 21 days repeated oral dosing (500 mg/kg)**

Groups	Hb (gm/100ml)	RBC (millions/cu.mm)
Control	14.45±0.4113	5.20±0.047
Test	15.67±0.4082	5.210±0.12 <sup>ns</sup>

N=6; Values are expressed as mean ± S.D followed by Students Paired ‘T’ Test

Ns – non significant when compared to control groups

**Effect of Siddha formulation (SN) on Biochemical markers of liver and kidney after 21 days repeated oral dosing (500 mg/kg/po) in rats**

Groups	ALP (K.A.Units)	AST (IU/L)	ALT (IU/L)	Urea (mg/100ml)	BUN (mg/ 100ml)	Glucose mg/dl	Cholesterol mg/dl
Control	2.973±0.3929	79.89±1.906	25.48±2.93	16.38±2.12	7.52±0.84	83.57±6.97	53.75±6.90
Test	3.233±0.427 <sup>ns</sup>	159.2±6.34 <sup>***</sup>	15.73±1.34 <sup>***</sup>	40.83±3.061 <sup>***</sup>	18.88±1.457 <sup>***</sup>	83.35±17.35	43.93±8.84 <sup>ns</sup>

N=6; Values are expressed as mean ± S.D followed by Students Paired ‘T’ Test

Ns – non significant when compared to control groups



### **Histopathological studies**

Animals were sacrificed at the end of repeated oral toxicity and tissues were processed for histopathological studies.

### **In Vivo Antioxidant study**

Samples of serum collected from rats treated with test drugs were assayed for GSH (Moron *et al* , 1979) and LPO (Yagi, 1976) and the results were compared with control group.

## **RESULTS**

### **Preliminary basic, acidic radicals and phytochemical studies**

The qualitative chemical analysis and acidic, basic radicals assay of the drugs showed the presence of phytoconstituents and minerals as depicted in (Table 1).

### **Acute oral toxicity study**

SN at the dose of 2000mg/kg/po did not exhibit any mortality in rats. As per OECD 423 guidelines the dose is said to be “Unclassified” under the toxicity scale. Hence further study with higher doses was not executed.

### **Repeated oral toxicity for 21 days**

Test drug SN at the dose of 500 mg/kg/po when administered orally for 21 days in rats did not show toxicity in Haematopoietic system (Table 2). However biochemical marker studies showed evidence of significant alteration in Liver function and kidney functions without any change in the serum glucose and cholesterol values (Table 3).

### **Histopathological study**

SN at the dose of 500 mg/kg/po daily administered for 21 days did not show evidence of pathological lesions in the tissues tested (Plate 1).

### **Antiinflammatory and Antipyretic studies**

SN at the dose of 500 mg/kg/p.o showed significant antipyretic activity and the drug also exhibited significant antiinflammatory activity in both acute (carrageenan hind paw edema) and chronic (cotton pellet granuloma) models of inflammation in rats. The results of SN in both models can be compared to that of standard drug diclofenac sodium (5 mg/kg/p.o) (Table 4 and 5).

### **Antioxidant activity**

At the end of 21 days repeated oral toxicity study when the plasma of drug treated animals was examined for GSH activity, the level of GSH activity was increased significantly ( $p > 0.001$ ) in test groups. On the other hand the LPO activity was considerably reduced in drug treated group when compared to control (Table 6).

## **DISCUSSION**

The reverse pharmacological and toxicological profiles of the siddha formulation SN was studied in experimental rats. The objective of the study was to prove/disprove the correlation of response for the treatments with the formulation in signs and symptoms of inflammation in Kalanchaga Padai (Psoriasis)

The drug was proved to be non toxic upto 2000 mg/kg/p.o in acute toxicity study. As per OECD 423 guidelines the drug is “Unclassified” in the toxicity scale. Hence further study with higher doses was not undertaken.

The phytochemical analysis of the formula revealed the presence of traces of flavonoids, saponins, chloride and sugar.

After 21 days repeated dosing with the drug (500 mg/kg/p.o) the animals did not show signs of toxicity in Haematopoietic system, food and water intake, weight etc. However the repeated dosing with drug for 21 days did exhibit alteration in liver function tests (ALT and AST) when compared to untreated controls. The drug also elevated the blood urea of rats without affecting the Blood Urea Nitrogen (BUN). Serum glucose and cholesterol levels remained unaltered in treated animals.

The test drug exhibited significant antipyretic and anti-inflammatory activity in both acute and chronic models of inflammation in rats. The drug at the dose of 500 mg/kg/p.o significantly effected both the exudative and proliferative stages of inflammation and the activity can be comparable to Standard Non Steroidal Antinflammatory (NSAIDs) agent Diclofenac Sodium (5 mg/kg/p.o). Since significant reduction in paw edema volume was observed at the end of 4<sup>th</sup> hour after carrageenan, the mechanism of action can be attributed to the inhibition of cyclooxygenase enzymes (COX), a similar mechanism established to standard drug, Diclofenac Sodium.

The results of the present study can be summarized as follows :

1. The formula SN is relatively non toxic upto 2000 mg/kg/p.o, which is “Unclassified” in the toxicity scale according to OECD 0 423.
2. In the repeated oral toxicity study (500 mg/kg/p.o) for 21 days no significant changes were observed in Haematological parameters, blood sugar, cholesterol, body weight, food and water intake and behavioral parameters of drug treated animals.
3. Liver function tests (LFT) did exhibit significant alteration in AST and ALT levels after repeated dosing for 21 days which do not have any correlation with clinical study with the drug administered for 48 days. The test drug did not exhibit any alterations in the normal architecture of the liver at the end of 21 days. Since there is no report on the LFT done in clinical protocol of study, it can be reasonably assumed that the drug is safe for humans unless and otherwise proved with clinical data generated on LFT. Blood urea level was also increased in the repeated oral dosing of the drug in animals. However, no evidence of liver or kidney toxicity in the histopathological study of the respective tissues at the end of 21 days repeated oral dosing. The mechanism(s) of these alterations are to be investigated further. However clinical study reports show no evidence of altered kidney function tests in patients treated for 48 days.

4. There is a good correlation of data on anti-inflammatory activity in animal and clinical studies.
5. In the above circumstances, monitoring of patients for liver and kidney functions in the long term treatment with the drug should be made mandatory in clinical practice.
6. The formulation exhibited significant anti oxidant and inhibition of LPO in rats treated for 21 days.



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